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MEDICAL

Vol. 35 MAY, 1954 No. 5

SURGERY

A Monthly Journal Devoted to the Art and Science of Surgery

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Vol. 35, No. 5, May, 1954. Surgery is published monthly by The C. V. Mosby Company, 3207 Washington Blvd., St. Louis 3, Mo. Subscription price: United States, $14.00 a year; Canada, $14.50; Foreign, $15.50. Entered as Second-Class matter January 20, 1937, at the Post Office at St. Louis, Missouri, under the Act of March 3, 1879. Printed in the U. S. A.

SURGERY

VOL. 35 MAY, 1954 NO. 5

Original Communications

SURGICAL CORRECTION OF AORTIC INSUFFICIENCY

CHARLES A. HUFNAGEL, M.D.,* W. PROCTOR HARVEY, M.D.,**
PIERRE J. RABIL, M.D.,*** AND THOMAS F. MCDERMOTT, M.D.****
WASHINGTON, D. C.

(From the Georgetown University Medical Center)

THE treatment of aortic insufficiency has always offered a serious problem in medical management once the signs of failure have appeared. Like many of the other acquired valvular diseases, the onset of failure is to a large measure the end result of the mechanical defect, which after a prolonged period of time imposes an insurmountable strain upon the myocardium. This, in effect, is a reflection of the excessive work-load imposed by free insufficiency. A reduction or complete correction of the valvular defect would, therefore, offer great hope for these otherwise incapacitated patients. Ideally, such correction of the valvular defect, if instituted early, might prevent the development of myocardial damage, or, if achieved later, might permit the changes which have occurred to be reversed.

The surgical treatment of aortic insufficiency has been extensively investigated in experimental animals. In our laboratory and in others, attempts have been made to reconstruct the aortic valve.[1-3] While many of these experiments have shown early promise, the late results have not appeared sufficiently good for application in the clinic. Other attempts have been made to replace the aortic valve with transplanted tissue. In most cases this transplanted tissue has been a homologous transplant of either the aortic or pulmonary valve as a unit. Again, the late results have been less satisfactory than had been hoped. Still other methods have been devised to reconstruct a valve with segments of pericardium, veins, arteries, or a combination of these structures. While some of these methods may be useful ultimately, we have felt that they have been distinctly less satisfactory than another line of endeavor which we have been pursuing since 1946.

This research was supported by a grant-in-aid from the National Heart Institute of the National Institutes of Health of the United States Public Health Service (H-914).

Received for publication, Sept. 14, 1953.

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In 1944, an investigation of the feasibility of replacement of major portions of the arterial tree with plastic materials was begun. During the course of this study, in the past, a number of significant facts were established. It was found that properly designed tubes of methyl methacrylate could be used permanently to replace portions of the aorta. It was established that the surface properties of methyl methacrylate inhibited coagulation of blood, and this gave further evidence that the concept of Hirschboeck[4] was correct. It was postulated that nonwettability of the surface was related to inhibition of clotting, and that plastic materials other than methyl methacrylate having a similar property showed similar results, but the best results were obtained only when the surface was highly polished.[5] Later this concept of nonwettability was investigated further by Walter and Murphy,[6] who termed this property hemorepellency.

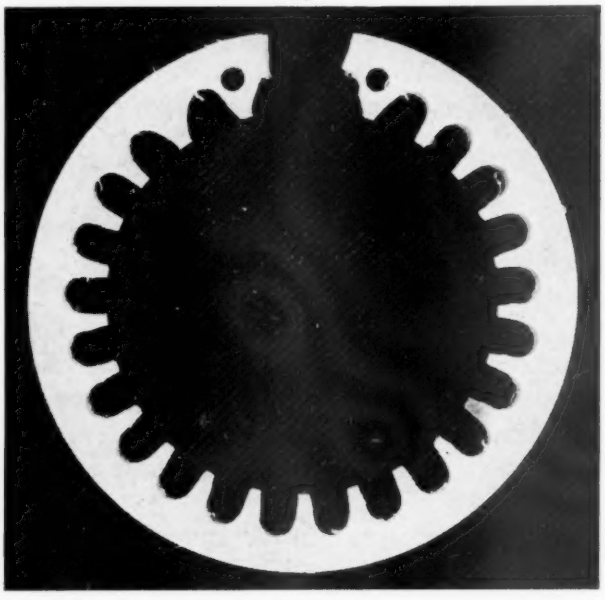
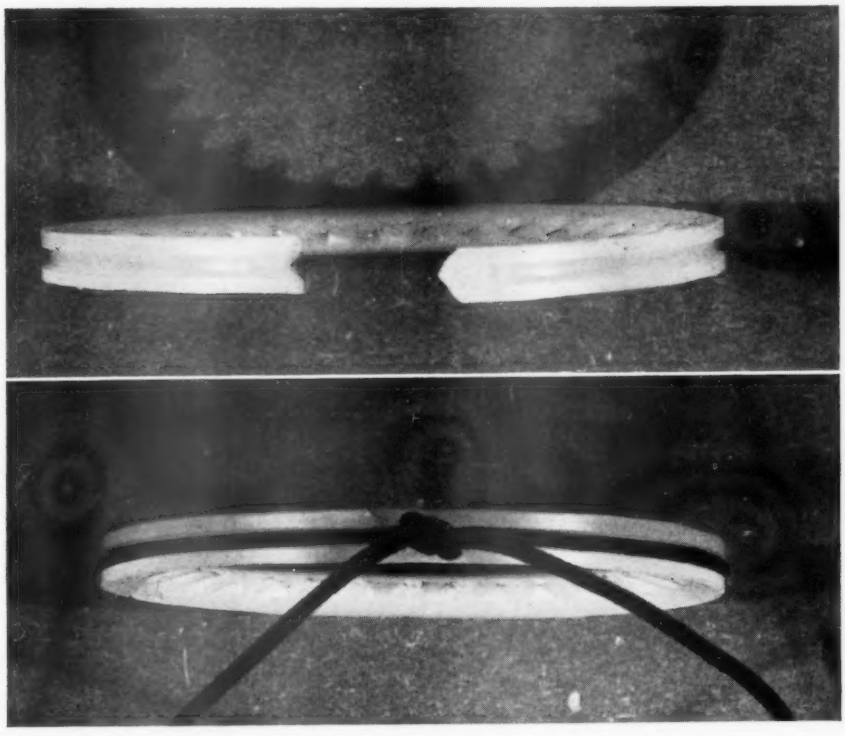


Fig. 1.—The details of the multiple-point fixation ring are shown. The openings for the ring-holder are adjacent to the split. The split allows the ring to be placed around the aorta before it is divided.

A further outgrowth of this early investigation was the development of the concept of multiple-point fixation.[5, 7, 8] In order to fix permanently a rigid prosthesis in the aorta, it was necessary to devise a method other than simple ligature. Many types of ligatures were tried in this investigation. Essentially, all of these which utilize continuous circumferential pressure for fixation produce necrosis of the aortic wall distal to the ligature. This uniformly produced fatal hemorrhage or aneurysm with delayed hemorrhage. In order to obviate this difficulty, continuous circumferential pressure was abandoned, and a device was designed which exerted pressure at discrete points on the circumference of the aorta while leaving intervening areas in which no pressure was exerted. We termed this multiple-point fixation. At the present time it is obtained using a nylon ring with multiple teeth projecting from its inner surface. The teeth are of sufficient length to elevate the body of the fixation ring below the outer

aortic surface. The teeth are sufficiently close together to coapt the aortic wall to the prosthesis without leakage between the projecting points. The ring is split at one point so that it can be opened and placed around the intact aorta. Other details of the construction of the ring are shown in Figs. 1 and 2. The small holes adjacent to the split serve a dual purpose. They are used to admit the points of a holding instrument which firmly close the ring around the prosthesis and permit immediate fixation of the aorta to the prosthesis when the instrument is closed (Fig. 3). After fixation is made permanent by tying the ligature which surrounds the body of the ring, the holding instrument can be withdrawn, and a stainless steel wire (Surgaloy) is passed through these

A.

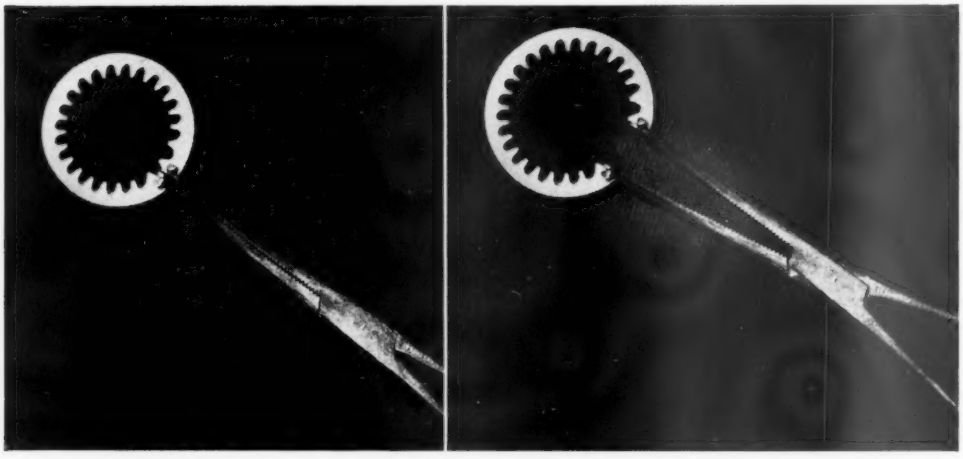


B.

Fig. 2.—The beveled construction of the split ring is shown in *A*. The closed ring with the tied ligature is shown in *B*.

holes and tied for further security. This method prevents the necrosis of the distal portion of the aorta, and is now used routinely to maintain the position of the prosthesis in the aorta. This type of fixation has been used experimentally several hundred times with no fatality caused by erosion or slippage of the fixation device. To date, it is the only method to be entirely satisfactory for the fixation of a rigid prosthesis in major vessels. It has been employed throughout this study and has been to a large extent responsible for the success of the permanent intubation method employed.

The plastic valve naturally evolved following the success of the permanent intubation of the aorta. The valve is essentially a modification of the tube. Many types of plastic valves have been employed experimentally. The majority of these have been some modification of a ball-type valve, although a number of flexible- and rigid-type valves have also been used. To date, the most useful



A. *B.*

Fig. 3.—The ring-holding instrument is shown in the closed position in *A* and in the open position in *B*.

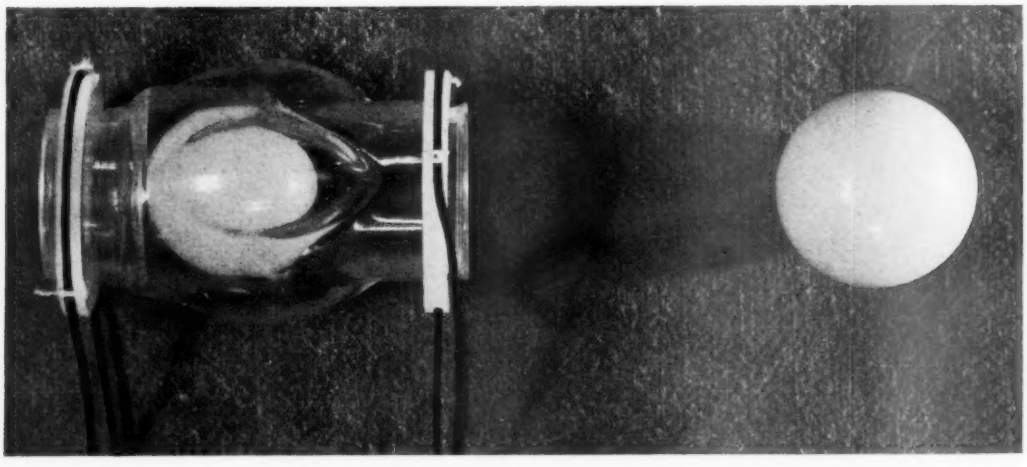


Fig. 4.—The assembled one-piece valve is shown with the multiple-point fixation rings in place in the grooves of the valve. The smooth surface of the polyethylene ball is demonstrated by the ball which is shown at the right before assembly in the valve chamber.

valve has been of the simple ball type which is shown in Fig. 4. Many modifications of this basic design were used before the development of this current design. The valve is composed of an inlet, a chamber which contains the ball, and an outlet. The entire valve is molded in a single piece so that the inner surface can be made extremely smooth and without seams. The ball itself which rests in the chamber has been made of methyl methacrylate and later of polyethylene. The design of the chamber is such that the ball is guided from the seat

to the valve stops in an essentially straight line. When the ball is constructed of methyl methacrylate, it is made hollow so that its specific gravity is slightly less than one. This ball is also made without an outside seam. Polyethylene balls are made solid since the specific gravity of polyethylene is quite satisfactory. However, in order to decrease further the weight of the ball, polyethylene balls can also be made hollow without external seams. The relation of the total cross-sectional area of the chamber is adjusted to prevent serious impedance to flow through the valve. Thus, under given pressure the flow through the valve is essentially equal to that which would flow through a simple tube of a diameter equal to the inlet of the valve. The valve will function in any position. The pressure differential necessary to open or close the valve completely is 5 mm. Hg. At each end of the valve there is a groove on the outer surface. This is immediately adjacent to the inlet and outlet ends. The grooves are sufficiently deep to hold the aorta and the fixation rings without slippage. The fixation rings and the grooves are matched so that there is a constant relation between the inner diameter of the ring and the groove. This allows for the passage of the aortic wall between the inner end of the teeth and the groove. The fixation ring is made of solid semiflexible nylon. The outer surface of the body of the fixation ring is grooved. The purpose of this groove is to maintain the position of the heavy braided silk ligature on the fixation ring. From the inner surface of the ring, the multiple teeth project. The body of the ring is split between two of the teeth. This split was made in the form of a V so that when the ring is closed the ends dovetail. Immediately adjacent to the split of the ring, two small holes are drilled through the body of the ring. These holes permit the insertion of an instrument to open and close the ring. The use of this instrument has the further advantage of producing immediate fixation of the prosthesis in the aorta when the instrument is closed. It also allows the fixation suture to be tied without tension.

A valve-holding instrument is also used in placing the valve. This instrument fits the contours of the valve and holds it firmly while it is being inserted.

This method, the prostheses, and the instruments employed have been developed through extensive experiments in the laboratory. During the course of these investigations such plastic valves have been placed into the thoracic aorta of 300 dogs for varying lengths of time. Some dogs are still living five years after the insertion of the valve. Others have been sacrificed at various intervals. When the valves have been made properly, we have had no instance of clotting. Imperfections of the inner surface of the valve, however, are very harmful. Failure to remove foreign substances such as wax, grease, or other materials used in the manufacture may result in clotting in the small-size valves. However, after obtaining uniformly good results by the elimination of these variables, a series of patients with severe aortic insufficiency was selected for a clinical trial with this method. For clinical use the valve* is made in four

*The valves and fixation rings are manufactured by the Brunswick Manufacturing Company, Boston, Mass. We wish to acknowledge the extremely helpful cooperation of Mr. Carl Hewson, whose continuing efforts have made possible the production of this valve.

We wish to acknowledge the assistance of Dr. Herbert Broida of the Bureau of Standards and Mr. Phillip Pfaff of the Bureau of Standards for their many efforts and suggestions in the investigation and study of the valve, and in particular Mr. Pfaff's help in the design of the ring-holding device.

sizes, ranging from three-quarters inch to one and one-eighth inch in inside diameter. Each size is one-eighth inch larger than the one smaller. It has been our experience that such variation in size is essential to meet the requirements of the different diameters in the human aorta. We have employed each of these sizes in patients.

PROCEDURE

The descending aorta just beyond the left subclavian artery has been chosen for the site of the valve for several reasons. In this site it is not necessary to occlude total blood flow, and this greatly reduces risk of operation in critically ill patients. In this site the aortic occlusion is well tolerated for rather prolonged periods so that the margin of safety is great; also, approximately 75 per cent of the total regurgitant flow is controlled in this site and the dangers of air embolization are minimized. We feel that this site offers the ideal relationship between maximum benefit and minimum risk.

The left chest is opened through the bed of the fifth rib, and the aorta is freed from its bed from the base of the left subclavian artery over a distance of 6 to 8 cm. The intercostal vessels are ligated and divided. The nylon rings together with their attached ligatures are passed around the intact aorta. The closing instrument is then inserted into each of the nylon rings. The valve is carefully inspected and washed with saline solution. These valves have been sterilized by soaking for eighteen hours in 1:500 Zephiran, and triply rinsed in sterile saline solution prior to being placed on the operating table. The valve holder is then applied to the valve so that it is prepared for insertion. The aorta is then occluded with Potts aortic clamps, and the aorta divided. A segment of aorta is removed. The distal end of the aorta is then grasped at equidistant points in Judd-Allis clamps and held open widely. The valve is inserted into the opened aortic end, and the ring closed into the groove with the holding clamp. The ligature is then tied. Great care is taken to see that the ring fits well and that the edges of the ring are well approximated when the ring is closed. The ring-holding clamp is removed. The Judd-Allis clamps are applied to the proximal aorta. The proximal end of the valve is inserted, and the ring similarly closed and tied. The ring clamp and valve holder are then removed. The valve is filled with saline solution containing 1 mg. of heparin per 10 c.c. As much air as possible is removed from the valve. The distal aortic clamp is then removed, and following this the proximal clamp is removed. These clamps are taken off slowly, and blood flow to the aorta is now resumed. A twisted Surgaloy No. 000 suture is placed through the holes in the ring and tied. This gives further security to the valve and to the ring, insuring against any slippage, giving additional support in case the ligature should break. The same process is repeated at the distal end. Immediately following the removal of the aortic clamps, the valve can be heard to open and close. A pleural flap is then turned down from the lateral chest wall and sutured over the valve after a small square of Gelfoam is placed between the valve and vertebral bodies. After the pleural flap has been sutured in place, the valve is covered completely. The chest is then closed in the usual fashion.

Since September, 1952, twenty-three patients have had this type operation for aortic insufficiency, using the currently acceptable valve and multiple-point fixation.[9] Most of these patients have been in the terminal stages of their disease. Actually, all have shown signs of cardiac failure, ranging from mild to severe. All have had diastolic gallop rhythms and Austin Flint murmurs associated with their severe aortic insufficiency. In almost all of these patients the cardiac enlargement has been extreme. In several instances, technical difficulties were encountered because the heart filled almost the entire left chest. Three patients have had clinical and x-ray evidence of mitral stenosis and have had exploration of the mitral valve at the same procedure. We have attempted to select patients whose predominant lesion was that of aortic insufficiency, with the exception of the three cases listed previously. All of the patients selected have had extremely wide pulse pressures, none having a diastolic pressure higher than 40 mm. Hg. Many of the patients have had angina pectoris. Further evidence of the severe disease in most of these patients is shown by conduction defects which were present in almost all. Liver disease secondary to cardiac disease has been common. One patient has had complete coarctation of the aorta associated with severe aortic insufficiency, and both lesions were corrected at the same procedure.

These patients have been classified using a grading similar to that used clinically for mitral stenosis, with the necessary allowances for the differences between aortic and mitral disease. All except four of these patients have been Class IV, the remainder Class III. It is interesting that in a patient* with a coarctation of the aorta, it was not possible to place the valve into the stump of the aorta since the area of coarctation extended to the take-off of the left subclavian artery. This made fixation of the valve in this location extremely difficult, if not completely impossible. Therefore, a portion, two inches long, of the distal aorta was dissected free and removed as a free aortic segment. This was then transplanted into the proximal aortic stump by direct suture technique. The valve was placed between this graft and the distal aorta. In essence, this constituted an autogenous aortic graft. This represents what we believe to be the first instance in which such an autogenous aortic graft has been placed in the human being. This patient has done extremely well postoperatively.

Anesthesia is a serious problem in such patients who are already suffering from conduction difficulties and who are, in the normal course of their disease, prone to sudden death. Two patients not included in this series were offered operation and died during induction of anesthesia before operation had begun. A technique has been developed† for induction and maintenance of these patients on an extremely light anesthesia, which has been most encouraging and which represents a major achievement in the control of these patients.

Among the first ten patients, there were four deaths. In the second series, Table I shows the distribution and the cause of death in the various categories. In the first group of ten patients, there was one death on the operating table

*Kindly referred to us by Dr. H. Shumacker.

†By Dr. Thomas McDermott, professor of anesthesiology, Georgetown University Medical Center.

of a terminal patient who had been bedridden for the past year. He had multiple decubiti and other evidences of severe debility. Death occurred during operation, but prior to the insertion of the aortic valve. This patient represents one in whom the operative procedure was not feasible because of the late stage of disease. Operation was offered to this patient as a last resort. One patient succumbed to subacute bacterial endocarditis some six weeks following operation. This apparently had been present preoperatively and had been in part responsible for his rapid decline prior to operation, although repeated blood cultures had been negative. The diagnosis was made at post-mortem examination. The patient had been treated with multiple antibiotics in large doses, all without results. One patient in this first group also died of an intractable pneumonitis, which was a causative agent in the onset of ventricular fibrillation in a patient with a severely impaired conduction system. This patient died several days after operation. One other patient succumbed ten days after operation, apparently of an acute cardiac arrhythmia. Autopsy showed no anatomic causes. Except for the death on the operating table, these deaths represent essentially complications of underlying disease.

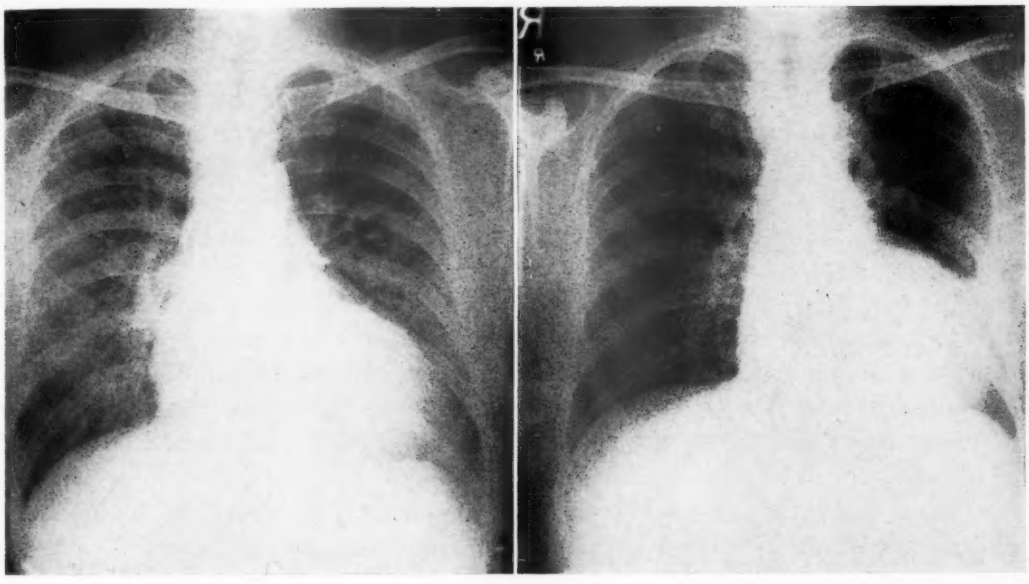
TABLE I

	TOTAL	OPERATIVE MORTALITY	POSTOPERATIVE MORTALITY	LIVING AND IMPROVED
Series I (10 cases)				
Class III	1	0	0	1
Class IV	9	1 cardiac standstill	3: arrhythmias, 2; subacute endocarditis, 1	5
Series II (13 cases)				
Class III	3	0	0	3
Class IV	10	0	2: myocardial failure, 1; acute rheumatic fever, 1	8
Totals	23	1	5	17

In the second series of patients, one patient died of congestive failure several days postoperatively. This was another patient who was treated rather late in the course of disease with great difficulty in the control of failure. The second patient died of what appeared to be an acute exacerbation of rheumatic fever following operation associated with other complications not directly related to operation. We have thought that the most common cause of death in the postoperative period has been ventricular arrhythmia, and this has always been associated with serious degrees of conduction difficulties known preoperatively. We feel that patients with such conduction defects offer a very serious risk in contrast to those in whom there has been no serious change in rhythm.

There has been no case to date in which there has been evidence of failure of the valve to function or of clotting. The pulse contours in the lower extremities have become normal while those in the upper extremities have remained Corrigan in type. Electrocardiograms have shown no evidence of myocardial ischemia after or during operation, except as it existed prior to operation. Murmurs which have been present preoperatively have diminished after operation. There has been an increased exercise tolerance in all patients and a great decrease or

absence of the necessity of mercurials. In all surviving patients there has been a dramatic improvement, which has been associated with a reduction in cardiac size as measured by x-ray film, as shown in a representative case in Fig. 5. This change in cardiac size can be noted in the first six weeks after operation. The rate at which this continues is not fully established because of the relatively short duration of the follow-up. It is believed that this decrease in cardiac size is due to decrease in dilatation of the left ventricle. In some patients there have been certain systolic murmurs at the apex which have been clinically interpreted as mitral insufficiency. No evidence of organic mitral insufficiency, however, could be found on exploration or on palpation of the auricle at operation. This type of murmur has diminished in relation to cardiac size.



A. *B.*

Fig. 5.—*A* shows preoperative x-ray film of heart. Patient had an Austin Flint murmur and a diastolic gallop. *B* shows same patient one month after operation.

DISCUSSION

It is interesting to note that in the patients who comprise this series, the vast majority of cases of relatively pure aortic insufficiency are of rheumatic origin. Syphilitic disease has constituted only about 20 per cent of this group. The age of the patients has varied from 17 to 57 years. This is in sharp contrast to the common concept that rheumatic valvular involvement is predominantly that of aortic stenosis with little or no aortic insufficiency. One patient with syphilitic aortitis had an extremely thick aortic wall, which presented serious technical problems. However, it was possible to insert the valve and utilize the multiple-point fixation technique. Similarly, in the older age group of patients in whom there has been arterial atherosclerotic disease concomitant with the rheumatic process the use of multiple-point fixation has been most satisfactory.

The valve has been inserted into the descending aortic arch for several reasons. At this level there is a relatively long period of safety in which the aorta can be occluded without serious difficulty in the normal patient. We consider twenty minutes to be the maximal safe period. The actual insertion of the aortic valve usually requires between three and six minutes. The total period of occlusion in actual practice is within this range of time. In this location, approximately 75 per cent of the reflux is controlled. Attempts to place it in a more physiologic position were met with extreme difficulty. Technical difficulties which occlude the blood supply to the brain during insertion of the valve are very real. Air embolization by failure to eliminate all of the air from the valve is another serious problem. From techniques for deviation of the circulation, it would certainly be possible to place this valve in the ascending arch. On the basis of evidence from certain animal experiments which are not reported in detail here, we are questioning the validity of placing the valve in this location even if it were simple technically. However, we feel very strongly that these patients are in an extremely precarious physiologic state. Major manipulations near the heart and the great vessels are extremely hazardous. Attempts to explore the aortic valve during operation are always associated with serious arrhythmias. We feel that placing the valve in the aortic arch makes for minimal risk to the patient while maintaining almost all of the benefits by correcting the great majority of regurgitation.

In patients with combined aortic stenosis and insufficiency, it is very reasonable to suppose that by first placing the aortic valve in the aorta one might then proceed with an aortic valvulotomy. We have not encountered a patient in whom this appeared to be indicated; however, in the correction of aortic stenosis the dangers of producing insufficiency might be greatly minimized by the use of the valve.

After the valve has been inserted in the patient, the sound of the opening and closing of the prosthesis is readily audible, using the stethoscope. During the first few days postoperatively, the sound may be audible to the unaided ear. Patients are informed of this prior to operation. It has given rise to no difficulties in any patient to date. After the passage of several weeks to months, the sound gradually diminishes. This apparently is associated with covering of the valve with fibrous tissue. Proximity of the valve to the left main-stem bronchus always makes the valve audible if one listens to the patient who has the mouth open in a very quiet room. It is rather striking to note that most patients volunteer the information that while, prior to operation, they had the sensation of great throbbing throughout their entire body with each heart beat, and that at night the bed would usually pulsate with each cardiac contraction, this no longer occurs following operation.

CONCLUSION

Seventeen patients are now living and well following the insertion of an aortic valvular prosthesis. These patients have now been followed for periods of up to eleven months. These patients are greatly improved by the subjective and objective criteria. Currently, other types of prostheses are under study and

further investigations as to the optimum site for the insertion of a valvular prosthesis are still under way. However, this group of patients represents the first attempt at the surgical correction of aortic insufficiency in patients. The results are extremely encouraging at this time.

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TEMPORARY AORTIC OCCLUSION IN ABDOMINAL SURGERY

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(From the Department of Surgery, Ohio State University Medical School)

DURING the past year we have been interested in the use of aortic occlusion as a means of facilitating surgical procedures within the abdomen, and have suggested its trial in uncontrollable hemorrhage. Since all the viscera within the abdomen ultimately receive their blood supply from the aorta, it would appear that even the most furious of arterial hemorrhages within the abdomen could be controlled by aortic occlusion just above the celiac level. It is also apparent that regardless of the risk of aortic occlusion it cannot be greater than that of uncontrolled hemorrhage. In a previous report, aortic and portal vein occlusion as a method of obtaining hemostasis during partial hepatectomy was presented.[1] We have since used occlusion just above the bifurcation for one hour to facilitate the procedure of pelvic evisceration with minimal bleeding and without any complications.

Except for otherwise fatal conditions (aneurysms, emboli, and so forth), occlusion of the abdominal aorta has received little attention. Furthermore, we have been unable to find detailed studies which would indicate how long and at what levels the abdominal aorta may be occluded safely. We wished to determine the answer to this question (Series 1), record the effects on the cardiorespiratory system (Series 2), study the effect on blood electrolytes (Series 3), and attempt to improve survival by physical and chemical means (Series 4). In the latter three phases of the problem we have restricted our study to the celiac level for lethal periods of occlusion. Routine autopsy and microscopic sections were taken of each animal upon sacrifice or death. In this study, 114 dogs were employed.

PROCEDURE

Series 1.—The maximum safe time the aorta could be occluded above the celiac axis, superior mesenteric artery, renal arteries, and above and below the inferior mesenteric artery was determined by occluding the aorta with a Potts coarctation clamp for varying lengths of time. The time intervals employed are shown in Table I. Sterile operative procedures were carried out in each of 70 dogs. No special methods were employed to prolong their survival.

Series 2.—(a) Carotid and jugular pressures, pulse, and respiratory rates were determined in 5 dogs during a forty-minute period of aortic occlusion above the celiac axis and for one hour after release of the clamp (Table II).

Received for publication, Aug. 24, 1953.

Readings were taken every ten minutes, and the arterial pressures were recorded with a 0 to 760 mm. Hg pressure Statham gauge. Venous pressures were measured with a 0 to 300 mm. Hg pressure Statham gauge. These gauges were connected to a BL-320 Brush amplifier, and the output of the amplifiers was recorded simultaneously on a Brush Model BL-202 double-channel oscillograph. The pressure heads were connected to the animal by heavy-walled transparent plastic tubing, the latter being filled with heparinized isotonic saline solution. Arterial pressures were recorded from the left carotid artery, and venous pressures from the right jugular vein.

TABLE I. EFFECT ON SURVIVAL OF AORTIC OCCLUSION AT DIFFERENT LEVELS (70 DOGS)

MINUTES OCCLUDED	ABOVE CELIAC		ABOVE SUPERIOR MESENTERIC		ABOVE RENALS		ABOVE INFERIOR MESENTERIC		BELOW INFERIOR MESENTERIC	
	N	S	N	S	N	S	N	S	N	S
5	2	2								
10	2	2								
15	2	2								
20	2	2*								
30	2	1								
40	4	0								
60	4	0	2	2*	4	4*				
75			2	1	4	3				
90	4	0	2	0	4	1				
120			2	0	4	2				
150					4	2				
180					4	1	2	2*		
240							2	1		
300							2	1		
Ligation							5	0	5	3

N = number of dogs receiving aortic occlusion.
S = number of dogs surviving aortic occlusion.
*Maximum safe time.

TABLE II. MEAN VALUES DURING AND FOLLOWING FORTY MINUTES OF AORTIC OCCLUSION ABOVE THE CELIAC AXIS (5 DOGS)

	NORMAL	CLAMP ON	10 MIN.	20 MIN.	30 MIN.	40 MIN.	CLAMP OFF	10 MIN.	20 MIN.	30 MIN.	40 MIN.	50 MIN.	60 MIN.
Carotid pressure	155	181	207	211	206	196	96	120	116	113	115	114	110
	123	152	157	166	161	157	59	94	89	88	84	83	79
Pulse per minute	137	111	104	127	136	137	141	128	130	135	137	139	139
Respiration per minute	22	22	21	26	25	24	29	26	25	25	22	24	24
Venous pressure (jugular in mm. H_2O)	23.2	26.0	31.1	22.1	19.5	26.0	11.7	9.1	9.1	1.30	15.6	22.2	24.8

(b) In another group, 3 dogs were employed in which the celiac, renal, and femoral pressures were measured simultaneously with carotid blood pressure while the aorta was occluded at different levels. These levels were just above the celiac axis, superior mesenteric artery, renal arteries, and above and below the inferior mesenteric artery (Table III).

Series 3.—Blood chemistries were taken in 5 dogs following sixty to ninety minutes' occlusion above the celiac axis when the animals were in profound shock just prior to death.

TABLE III. THE EFFECT OF VARIOUS LEVELS OF AORTIC OCCLUSION ON CAROTID BLOOD PRESSURE WHEN MEASURED SIMULTANEOUSLY WITH CELIAC, RENAL, OR FEMORAL PRESSURE; EACH SECTION REPRESENTS AN AVERAGE OF THREE DOGS

	NORMAL	BELOW INFERIOR MESENTERIC	ABOVE INFERIOR MESENTERIC	ABOVE RENALS	ABOVE SUPERIOR MESENTERIC	ABOVE CELIAC	CLAMP OFF	1 MIN. LATER
Carotid	123	134	130	145	175	188	66	136
	86	95	88	95	119	137	47	92
Celiac	106	123	118	130	132	1	115	116
	77	87	78	83	104		69	86
Carotid	141	146	132	145	159	189	78	139
	107	112	99	114	130	155	49	99
Renal	130	142	131	18	9	9	122	119
	94	101	92	0			90	85
Carotid	153	147	142	156	168	184	85	138
	107	100	96	113	128	121	52	99
Femoral	156	21	22	13	23	16	136	134
	101						85	99

Series 4.—(a) To determine whether or not cooling of the liver would aid in prolonging survival, the liver was cooled by means of rubber-covered copper tubing placed around the organ. A refrigerant solution of alcohol and acetone was cooled by passing it over dry ice and through the copper tubing by means of a pump. The liver temperature of the 9 dogs employed was kept between 10° and 20° C. by changing the speed of the pump. The aorta was clamped above the celiac axis for varying lengths of time, as recorded in Table IV.

TABLE IV. EFFECT OF AORTIC OCCLUSION ABOVE CELIAC AXIS ON SURVIVAL WHEN THE LIVER IS COOLED AND DRUGS ARE ADMINISTERED (31 DOGS)

MINUTES OF OCCLUSION	COOLED LIVER		DIBENZYLINE (MG./KG.)										AUREOMYCIN		ALLOXAN		VITAMIN E	
			1 MG.		3 MG.		4 MG.		5 MG.		6 MG.							
	N	S	N	S	N	S	N	S	N	S	N	S	N	S	N	S	N	S
40			1	0	2	2*	2	2*	1	0	1	0	1	0	1	1*	1	0
50			1	0	2	0	2	0	1	0	1	0	1	0	1	0	1	0
60	3	3*											1	0			1	0
75	2	1																
90	2	0																
120	2	0																

N = number of dogs receiving aortic occlusion.
S = number of dogs surviving aortic occlusion.
*Maximum safe time.

(b) Dibenzyline* in varying doses (1, 3, 4, 5, and 6 mg. per kilogram) was given intravenously to 14 dogs thirty minutes before occlusion of the aorta above the celiac axis. Aureomycin was given intravenously (30 mg. per kilogram) in another group of 3 dogs. Alloxan in doses of 15 mg. per

*Courtesy of the Smith, Kline & French Laboratories.

kilogram was given to 2 dogs thirty minutes before aortic occlusion above the celiac axis. Vitamin E was also tried on 3 dogs (1 Gm. intravenously). The length of occlusions is given in Table IV.

DATA AND DISCUSSION

Series 1.—It may be seen from Table I that the maximum safe time of occlusion just above the celiac axis is twenty minutes; the superior mesenteric artery, sixty minutes; above the renal arteries, sixty minutes; above the inferior mesenteric artery, three hours. It is apparent from the difference in the safe time of the celiac and renal series that it is safer to occlude below the celiac axis than above it, and that the organ probably responsible for this difference is the liver. It would appear that the kidneys are not the limiting factor at the celiac level, as the safe time at the renal arteries is one hour. In addition, survivals were observed when the aorta had been occluded just above the renal arteries for as long as three hours, indicating that there is considerable variability in the amount of kidney damage produced by occlusion. Hind limb paralysis was noted sporadically when the aorta was occluded longer than one hour above the renal arteries. When this occurred the animals always expired within a few days. It is interesting to note the survival time following occlusion. The dogs which expired following occlusion above the celiac axis did so within twelve hours. In the renal series, the dogs which expired did so on the third to fifth day. Bloody diarrhea, vomiting, and anuria appeared in the latter group, and were of grave prognostic significance.

Since the safe time above the inferior mesenteric artery was so long (three hours), it was decided to divide the aorta above the inferior mesenteric in 5 dogs. This was followed by 100 per cent mortality, with findings at autopsy of a gangrenous rectosigmoid and urinary bladder in each case. However, when 5 dogs had a ligation and division of the aorta just below the inferior mesenteric artery, the mortality was reduced to 40 per cent. A necrotic urinary bladder was found in the dogs which expired.

The microscopic findings in the animals which died following occlusion of the aorta above the celiac axis may be briefly summarized as focal necrosis of the liver with little evidence of renal damage. In the animals which died following occlusion just above the renal arteries, no liver damage was noted; however, renal damage strongly suggestive of lower nephron nephrosis was observed.

Series 2.—The average values of blood pressure, pulse, respiration, and venous pressure may be seen in Table II during and following forty minutes of occlusion of the abdominal aorta, just above the celiac axis.

The effects of thoracic aortic occlusion on blood pressure, pulse, respiration, and venous pressure were noted as long ago as 1886.[2] However, the cardiovascular response as affected by the duration of the occlusion has not been found, nor have the effects of systematic clamping at various levels within the abdomen. Our findings are similar to those obtained when the thoracic aorta is occluded, namely, a rise in carotid blood pressure followed by a fall upon release of the clamp.[2-5] Most important is the failure of blood

pressure to reach the normal pressures at one hour. We have repeatedly observed marked hypotension prior to death. Pulse rate decreased markedly on application of the clamp above the celiac axis, and increased shortly after the removal of the clamp. This is probably due to the effects of the carotid and aortic sinus reflexes. Respiration and venous pressure were variable. It may be seen from inspection of Table II that venous pressure increases temporarily on application of the clamp and falls suddenly on removal.

The average values for the blood pressures measured in the celiac, renal, and femoral arteries simultaneously with carotid arterial pressures can be seen in Table III. The effect of clamping the aorta at higher and higher levels was a progressive increase in the carotid blood pressure. The same effect was noted in the renal and celiac arteries, except when the aorta was occluded above them, which resulted in a marked fall in blood pressure (Table III).

Series 3.—Blood chemical determinations in 5 dogs receiving aortic occlusion above the celiac axis for sixty to ninety minutes were made before occlusion and shortly before the animals expired. Comparing the postocclusion chemistries with the dogs' predetermined normals indicates no change in the cephalin flocculation time, thymol turbidity, total protein and albumin-globulin ratio, and prothrombin time. A moderate rise in the blood urea nitrogen and fall in CO_2 volumes per cent accompanied by a slight rise of potassium were noted in 2 dogs. It was our impression that these blood chemical changes were not of sufficient magnitude to account for the deaths resulting from occlusion above the celiac axis.

Series 4.—(a) At the present time, cooling the liver appears to be the most promising method of increasing the safe time of aortic occlusion above the celiac axis. It should be noted, however, that occlusion above the celiac axis cannot, even with cooling, be prolonged beyond one hour (Table IV). Sacrifice, at one month, of each of the survivors showed no gross changes, and microscopic sections showed minimal changes of the liver and kidneys.

(b) The effects of drugs in prolonging the maximum safe time of occlusion above the celiac axis may be seen in Table IV. Dibenzyline is seen to give protection in 3 to 4 mg. per kilogram doses, increasing the safe time to forty minutes of occlusion above the celiac axis. Alloxan appears promising, but Aureomycin and vitamin E were not effective in providing protection against the effects of aortic occlusion in the dose employed (Table IV).

It should be stated that the exact mechanism of death following aortic occlusion above the celiac axis is unknown. It seems clear, however, that the liver and kidneys play a predominant role in the outcome following aortic occlusion and that hypotension results from prolonged aortic occlusion above the celiac axis. We suspect that the mechanisms outlined by Shorr and associates[6] are involved when the aorta is occluded above the celiac axis and that VDM is one of the factors leading to fatalities when the time of occlusion is too long.

CONCLUSIONS

1. The maximum safe time of aortic occlusion just above the celiac axis is twenty minutes, above the superior mesenteric artery sixty minutes, above

the renal arteries sixty minutes, and above the inferior mesenteric artery three hours (60 dogs).

2. The mortality rate of ligation and division of the aorta just above and just below the inferior mesenteric artery is 100 per cent and 40 per cent, respectively (10 dogs).

3. Following lethal occlusions of the aorta above the celiac axis, carotid blood pressure does not return to normal, and continues to decline until death (5 dogs).

4. There is a progressive rise of celiac, renal, and carotid arterial pressures when occlusion of the aorta approaches these vessels from below, and a prompt fall in pressure occurs when the occlusion site is above them (3 dogs).

5. Cooling of the liver (10° to 20° C.) allowed sixty minutes of occlusion above the celiac axis without fatalities (9 dogs).

6. Dibenzyline and alloxan appear to exert protection from the effects of occlusion above the celiac axis; Aureomycin and vitamin E did not offer lasting benefit in the doses employed (22 dogs).

7. The liver and kidneys appear to play a dominant role in determining the outcome following aortic occlusion. Microscopic evidence of focal liver necrosis and renal tubular damage occurs in lethal occlusions above the celiac axis (all dogs).

8. Blood chemistries just before death in fatal aortic occlusions above the celiac axis were not of sufficient magnitude to account for the deaths (5 dogs).

Acknowledgment.—We wish to express our appreciation to Dr. Mel A. Davis for his careful review of the microscopic sections and helpful suggestions.

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COMPARISON OF LATE SEQUELAE OF COMMON AND SUPERFICIAL FEMORAL VEIN LIGATIONS

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THE purpose of this clinical investigation was the examination of the physical consequences of ligation and division of the superficial and the common femoral veins of human beings. The clinical indications for the interruption of these veins are shown graphically in Fig. 1.

This study consists of observations of two groups of patients treated at the Wayne County General Hospital between 1944 and 1947. Between the first days of September, 1944, and January, 1946, thirty-one ligations of the common femoral veins of twenty-eight persons were performed (Series I).

From March 1, 1946, to Nov. 1, 1947, forty-three ligations of the superficial femoral veins of thirty-six persons were completed (Series II). All but three† individuals of both series were examined repeatedly until Nov. 1, 1952, or until they died. Consequently, the follow-up period of Series I is roughly eight years long and for Series II it is six and one-half years.

The selection of the site of ligation was not varied between Sept. 1, 1944, and April 1, 1945; the common femoral vein was always interrupted. Between April 1, 1945, and Jan. 1, 1946, the ligation and transection of the common femoral vein were performed only when clotted blood was found in it; when no clot was found in it, the superficial femoral vein was interrupted. After Jan. 1, 1946, only ligations of the superficial femoral systems were carried out, even though the clot extended into the common vein. When clotting extended proximal to the point of junction of the deep with the superficial vein, proximal aspiration was performed through an opening into the superficial vein. Afterward it was ligated above and below the incision and divided. Whenever free backflow was not attained after aspiration, the superficial vein was ligated and divided and anticoagulants were given.‡

The actual lengths of the observation periods are shown in Table I.

No operative deaths occurred in either series, although the operation was performed upon twelve persons while they were critically ill.

The age distribution of both series were statistically the same. The combined age distribution is shown in Fig. 2. Seemingly, thrombosis in the Wayne County General Hospital was somewhat associated with aging.

The ligations and divisions of the veins were performed through vertical incisions in order to minimize the transection of major lymphatic vessels and to provide adequate exposure. The femoral artery was meticulously avoided.

Received for publication, Sept. 19, 1953.

*Kankakee, Ill.

†The status of these three individuals as of Nov. 1, 1952, was determined by correspondence because change of residence made it impractical to see them.

‡In a number of instances when free backflow was not obtained, the hole in the vein was closed with sutures, and sole reliance was placed in anticoagulants; these cases are not included in this study.

TABLE I

YEAR	SERIES I: COMMON FEMORAL VEIN,* NUMBER OF PATIENTS	SERIES II: SUPERFICIAL FEMORAL VEIN, NUMBER OF PATIENTS
Original group	28	36
1 yr.	22	36
2 yr.	16	36
3 yr.	15	34
4 yr.	12	33
5 yr.	10	32
6 yr.	10	18
7 yr.	8	
8 yr.	7	

The progressive decline in numbers of individuals in Series I during the eight-year period is due to death; that in Series II is due to the same cause up to the sixth year. The difference in the number of patients between the five- and six-year periods of Series II is due to the fact that fourteen of the thirty-two had been operated upon between March 1 and Nov. 1, 1947, and therefore had not yet lived six years after the operation by Nov. 1, 1952.

*Number of patients of the series living at end of year.

It was never freed from its bed because of the impression that such a maneuver was a possible cause of the vasospasm observed in a number of instances prior to the beginning of this effort.

The sequelae of particular interest of the ligation of the superficial and common femoral veins were: (1) persistent brawny edema, (2) dermatitis and cellulitis, (3) varicose veins, and (4) ulceration.

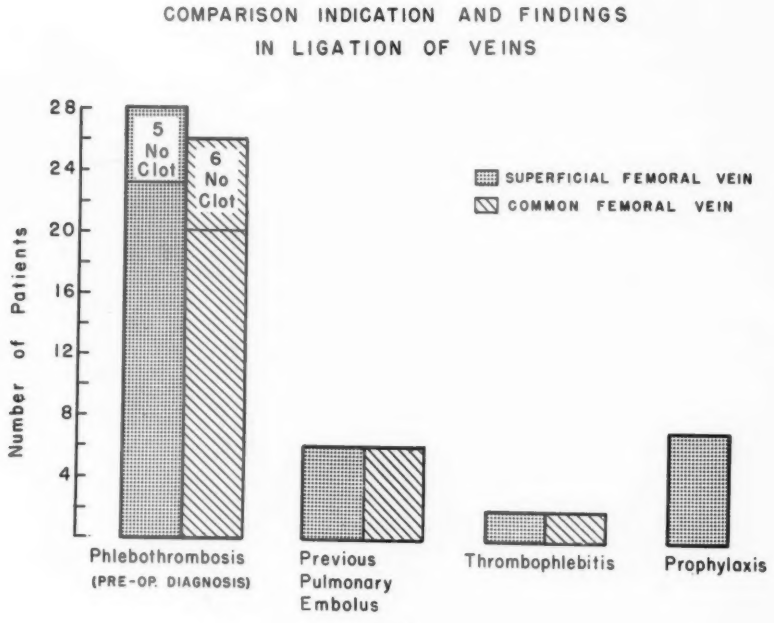

Fig. 1.

Table II contains the incidence of these sequelae at one, four, and eight, and one and five years for ligations of the common and superficial femoral veins, respectively. The percentile occurrences of edema, dermatitis, cellulitis,

varicose veins, and ulceration after ligation of the superficial and common femoral veins are shown in Figs. 3 through 6.

TABLE II. INCIDENCE OF SEQUELAE OF LIGATION AND DIVISION OF COMMON FEMORAL VEIN AND SUPERFICIAL FEMORAL VEIN

SITE OF INTERRUPTION	COMMON FEMORAL VEIN			SUPERFICIAL FEMORAL VEIN	
YEARS AFTER OPERATION	1	4	8	1	5
Brawny edema	19 of 22	10 of 12	6 of 7	2 of 36	2 of 32
Varicose veins	12 of 22	12 of 12	7 of 7	2 of 36	6 of 32
Ulceration	10 of 22	10 of 12	5 of 7	1 of 36	1 of 32
Dermatitis and cellulitis	6 of 22	5 of 12	7 of 7	2 of 36	2 of 32

DISCUSSION

The ligation and division of the common femoral veins of man are followed by an inordinately high incidence of undesirable physical phenomena relative to the incidence of the same obtaining after ligation and division of the superficial femoral veins. The probability that chance alone could account for the observed differences is so small (see Table III) that the difference between the incidences of complications in the two series is significant. These late complications of ligations of the common femoral vein are of sufficient magnitude to collectively prevent the self-support of most of the individuals suffering them (see Fig. 7).

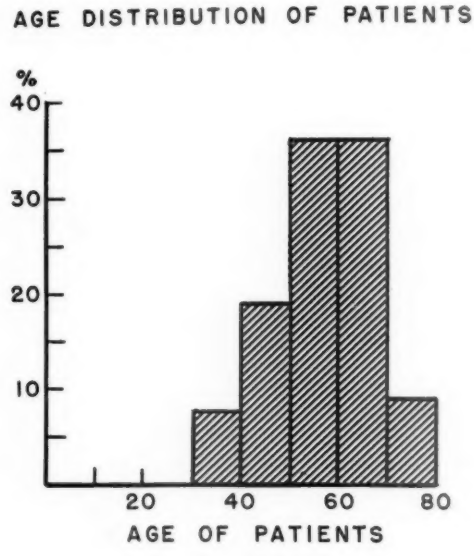

Fig. 2.

TABLE III. STATISTICAL COMPARISON

COMPLICATIONS	CHI SQUARE VALUES	PROBABILITY
Edema	26.000	< 0.001
Varicose veins	42.6297	< 0.001
Dermatitis	7.885	< 0.01
Ulcers	29.93946	< 0.001
Rehabilitation	18.0053	< 0.001

The reasons for the inordinately high incidence of late complications of ligation and division of the common femoral vein relative to that following the same procedures upon the superficial femoral system are not apparent.

Fig. 3.

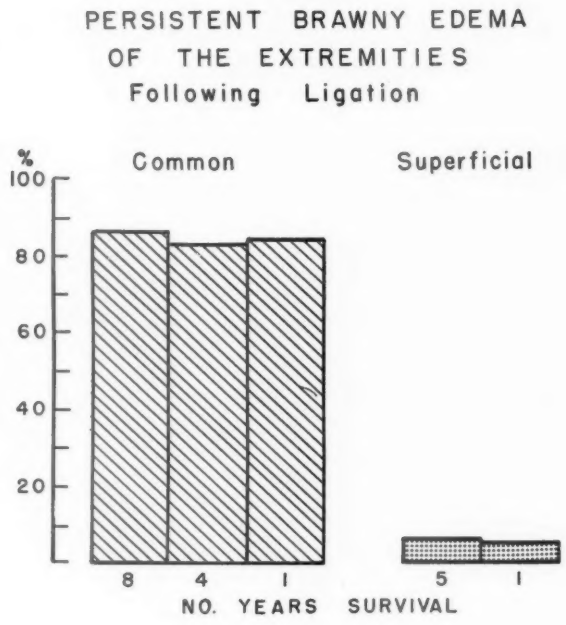

Fig. 4.

Conjecturally, the frequency and magnitude of the sequelae that follow the interruption of the deep veins of an extremity have been ascribed to the extent of the venous disease existing at the time of the ligation. A study of Table IV

leads to doubt concerning this concept, for, although the cases are few, they indicate that the site of the ligation of the deep venous system is more significant than the extent of the thrombosis prior to the ligation.

Fig. 5.

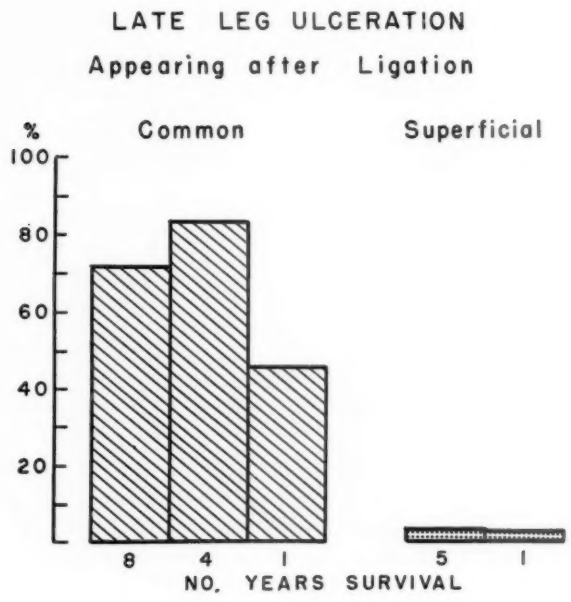

Fig. 6.

Although four patients of the six who had the common femoral veins interrupted when no clot was present in them died before one year had passed, all of them had developed varicose veins and brawny edema before they died. The two others suffered from all of the complications by the end of the first

postoperative year. On the other hand, one year after ligation and division of the superficial femoral vein in the absence of clot in the vein at the groin, none of twelve individuals had varicose veins, dermatitis, brawny edema, or ulcers, and only two of eight had varicose veins alone at the end of five years.

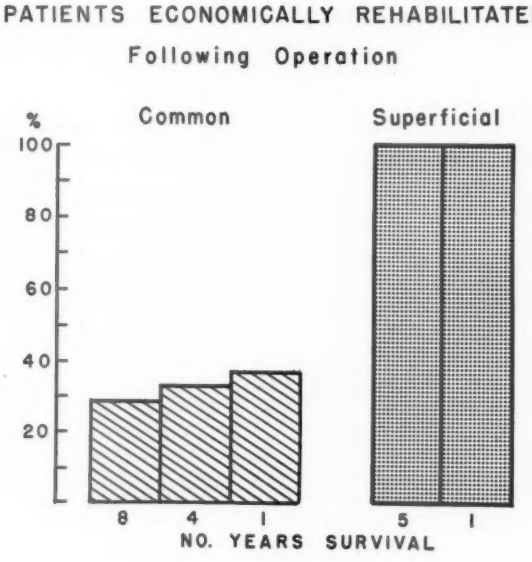

Fig. 7.

The temporal relationships between the various postligational sequelae are interesting in that they may be indicative of the etiological relationship of one to the other. Brawny edema appears first in practically all instances; nineteen of twenty-two persons showed it within one year after the ligation of the common femoral vein. Varicose veins tend to appear next, and dermatitis and ulceration follow. Postligational varicosities are not the sole factors in

TABLE IV. RESULTS OF BOTH TYPES OF LIGATIONS WHEN NO CLOT WAS PRESENT AT OPERATION

	COMMON FEMORAL VEIN			SUPERFICIAL FEMORAL VEIN				SUPERFICIAL FEMORAL VEIN			
	ERROR IN DIAGNOSIS			ERROR IN DIAGNOSIS				PROPHYLACTIC			
	NO. OF CASES AND YR.			NO. OF CASES AND YR.				NO. OF CASES AND YR.			
SEQUELAE	ORIGINALLY 6	ONE YR. 2	FOUR YR. 1	ORIGINALLY 5	TWO YR. 5	THREE YR. 4	FIVE YR. 4	ORIGINALLY 7	ONE YR. 7	THREE YR. 5	FIVE YR. 4
Edema		2	1		0	0	0		0	0	0
Varicose veins		2	1		0	0	1-2		0	0	1-2
Cellulitis, dermatitis		2	1		0	0	0		0	0	0
Ulcers		2	1		0	0	0			0	0
Economically rehabilitated		0	0		5	4	4		7	5	4

the etiology of brawny edema, ulceration, and dermatitis. All of the seven persons who lived eight years after the ligation and division of the common femoral vein had the acquired varices ligated and excised repeatedly, and yet five developed ulcers and six developed brawny edema. The chronic edema, ulceration, and dermatitis were checked to some extent, but not prevented or cured by removal and ligation of the varicose veins as they came. Therefore, the relationship of varicose veins to the other postligational sequelae is still speculative.

The causes of the hard edema which usually precedes ulceration are unknown. The hypertrophy of the epidermis, the subepidermal fibrosis and hyaline degeneration in it, the dilatation of the superficial lymphatic channels, and the hypertrophy of the superficial investing fascia of muscle which characterize the histologic picture of long-standing brawny edema are accompaniments of chronic lymphatic obstruction. However, no objective evidence for the existence of lymphatic stasis prior to the onset of brawny edema was obtained in these cases, so that no definitive statement can be made regarding the possible etiological role that might be played by postligational disturbances in lymphatic flow. Studies are now in progress in regard to this.

For a time, vasospasm was considered the primary etiological factor in the inception of the postligation syndrome. However, lumbar sympathectomy had no objective, long-term ameliorating, or curative effect on it. Between the second and fifth postoperative years, the lower lumbar ganglionic sympathetic chain was excised from six of the seven persons who were alive and who suffered from ulceration eight years after common femoral interruption. No prolonged improvement was noted in any case: universally the hard edema and ulcers recurred or persisted. Conceivably, in a few cases inadequate sympathectomy could have accounted for the failure to effect any prolonged beneficial influence on the postligational sequelae, but it is improbable that it could account for the universal failure. Therefore, it appears that vasospasm may be fairly safely discounted as the predominant cause of the edema, ulceration, and dermatitis following interruption of the common femoral vein. No transposition of the conclusions drawn from these observations to postphlebitic and varicose ulcerations should be made. The additional conditions imposed by ligation and division of the common vein may mitigate the possible effects of sympathectomy.

In so far as the selection of the site for the interruption of the deep venous system when indications for it exist, ligation of the common femoral system is to be avoided. A small personal experience* with individuals upon whom ligations of the inferior vena cava have been performed below the renal veins for extensive acute thrombotic disease with repeated pulmonary embolization provides us with the tentative conviction that ligation of the cava below the renal veins is less conducive to the induction of incapacitating complications than are ligation and division of the common femoral system.

*Three patients, all of whom have remained free of brawny edema and ulceration for four to eight years. All of them have extensive varices of both lower extremities.

Ligation and division of the superficial femoral vein can be performed with relative impunity.

CONCLUSIONS

1. Within one to eight years after ligation and division of the common femoral vein, incapacitating sequelae appear in more than three-fourths of the persons operated upon; however, ligation and division of the superficial femoral vein are attended by the same sequelae, with a frequency of less than one in ten.

2. Interruption of the common femoral vein is to be avoided.

3. Lumbar sympathectomy has no long-term curative influence on postligational ulceration, dermatitis, varicosities, or brawny edema.

4. The etiologies of the postligational sequelae are unknown. Apparently, the site of ligation is the primary determinant of ulceration, dermatitis, and brawny edema attending ligation and division of the deep veins of the lower extremity.

Acknowledgments.—We wish to thank Dr. M. Musselman and Dr. R. Gillespie of the Wayne County General Hospital, Eloise, Mich., for their cooperation in helping us to obtain part of the material used in this study.

AN EFFECTIVE CLINICAL METHOD FOR DETERMINING COAGULATION TIME OF BLOOD

Its Significance in the Presence of Intravascular Clotting

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THE coagulation time of blood is frequently utilized in the study of hemorrhagic diseases and in the evaluation of heparin therapy. Its reliability as a clinical test has often been questioned because of the variability of the results obtained. This dissatisfaction is manifested by the large volume of papers offering new techniques or modifications of others. Lee and White,[1] as early as 1913, citing Cohen, wrote, ". . . in a very complete paper on the 'Coagulation Time,' . . . (he) has discussed thirty-one methods and their modifications." To add to the confusion, no uniformity is found even among the descriptions of particular modifications of specific methods.

This report presents a method which is believed to be more effective clinically than previously reported ones. More elaborate ones are available, some utilizing electromechanical recording devices.[2] This method can be performed with simple apparatus in the laboratory, doctor's office, or at the bedside. It is practical and economical, and requires only such equipment as is already available in every clinic or hospital. It has been used by a variety of staff personnel under diverse conditions, including daily and room temperature variations, and has been found to give consistent, accurate results on like samples of blood.

A survey of the literature was first made, and several methods of coagulation time determinations were selected for trial. After a number of these proved inadequate in one respect or another, the method reported here was devised from several by incorporating portions which seemed desirable. This procedure, therefore, is not entirely original.

EQUIPMENT AND METHOD

The necessary equipment consists of five serially numbered, standard Wassermann tubes (12 by 75 mm.), a 10 c.c. Luer syringe, a small serologic tube rack, a 19 gauge needle, alcohol or acetone sponges, tourniquet, and stop watch.

All coagulation times were performed at room temperature with seasonal variations indoors ranging from 72° to 90° F. The syringe and needle were dry and sterile; the tubes were chemically clean, oven-dried, and allowed to at-

The study reported herein was supported in part by a contract from the Veterans Administration on recommendation of the National Research Council's Committee on Veterans Medical Problems.

The heparin used in this study was supplied by The Upjohn Company, Kalamazoo, Mich.

Received for publication, Sept. 8, 1953.

Fig. 1.

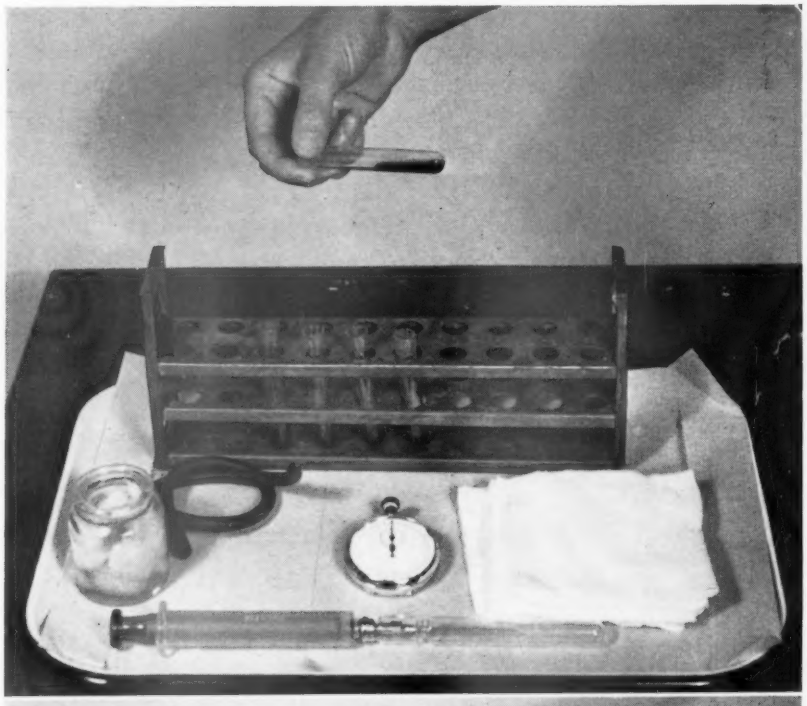

Fig. 2.

Fig. 1.—Photograph of necessary equipment and horizontally tilted tube.
Fig. 2.—Photograph showing point at which clotting is complete.

tain room temperature before being used. If the glass surfaces of any of the tubes or syringes which were to come in contact with the blood were etched, scratched, or broken, they were discarded.

The needle, with syringe attached, was inserted into a suitable arm vein after the usual preparation for venipuncture. Simultaneously with the appearance of blood in the syringe tip, the tourniquet was released and the stop watch was started. Aspiration was begun only after blood appeared in the syringe. Three to four cubic centimeters of blood was then rapidly, but gently, aspirated and the needle was withdrawn. Only those samples obtained by clean venipuncture were considered acceptable for the determination. If the syringe or needle was contaminated by tissue juice which contains thromboplastin, the blood sample was discarded, new equipment was obtained, and the procedure was repeated. Quick and associates[3] have stated that "even in taking blood by venipuncture enough tissue thromboplastin may occasionally gain entrance to reduce significantly the coagulation time, as Jacques and his co-workers have recently demonstrated."

The needle was removed from the syringe and 0.5 c.c. of blood was decanted carefully into each of the five glass tubes held upright in the test-tube rack. The tubes were filled in the reverse order of their intended examination (5, 4, 3, 2, 1).

Three minutes from the instant that blood first appeared in the syringe, Tube 1 was tilted from vertical to horizontal and replaced in the rack (Fig. 1). This process was repeated every thirty seconds until complete clotting occurred. The end point was defined as that time at which blood no longer ran along the surface of the horizontally tilted tube (Fig. 2). If the serum and cells separated into layers, the end point was defined as the time at which clotting became complete in all the layers. As soon as the end point was reached in the first tube, Tube 2, heretofore undisturbed, was tilted similarly every thirty seconds until its end point was reached. Serially, in like manner, the end points of the other three tubes were obtained. The end point of the fifth tube was recorded as the coagulation time of the blood.

CLINICAL STUDY

To determine the efficacy of the method, the study was submitted to clinical trial and statistical analysis of the data. Because the majority of thromboembolic diseases occur among hospitalized patients, both the diseased and control groups were selected from hospital patients. Several variables were thus eliminated. Control patients were on a similar diet, and the majority were in bed at least one-half of their waking hours. Excluded from the control group were patients recovering from operations, and those who had hepatic or renal disease, proved or suspected hemorrhagic diathesis, thrombosis or embolism (suspected or known), and those who were receiving or had recently received anticoagulants.

In many instances, duplicate examinations were made by different technicians on the same blood sample and on samples drawn at different periods. Daily variations and temperature effects were recorded. Among control pa-

tients with coagulation times of twenty to twenty-two minutes, the daily variation was less than one minute in repeated examinations. The variation between similar samples run in a water bath at 37° C. and those run at room temperature was one-half minute or less. In similar samples run by different technicians, it was never greater than one to one and one-half minutes.

These variations were considered of minor importance, since the error with commonly used methods is frequently as high as 10 per cent. Such slight deviations proved even less significant among the diseased patients, in whom the desired therapeutic range would be approximately three times normal, or pretreatment, coagulation time. If, with adequate dosage of heparin, the coagulation time of a patient whose pretreatment time was twenty minutes should be increased to approximately sixty minutes, a variation as great as three minutes would give an error of only 5 per cent.

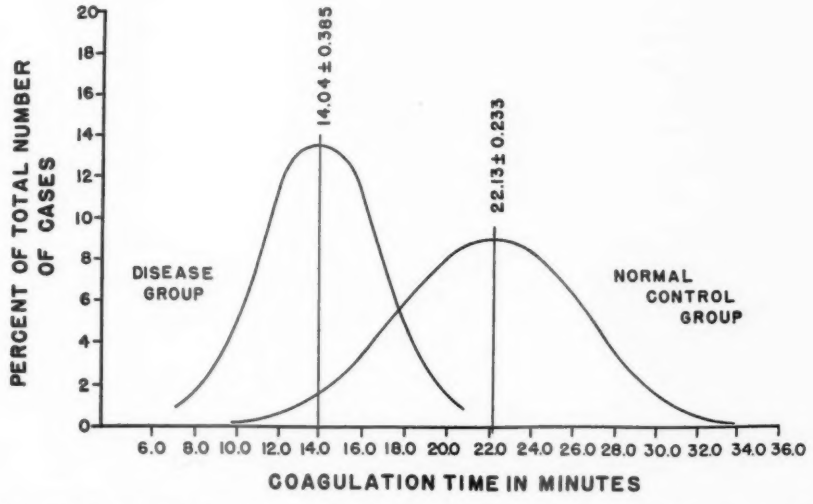

Fig. 3.—Graph showing variation in clotting times of patients in diseased and control groups.

After the method was standardized, coagulation times were obtained on 150 control patients. The results ranged between nine and thirty-nine minutes. The majority, 146, fell within a range of fourteen to thirty-two minutes, with 129 (86 per cent) showing clotting times of nineteen minutes or more. The mean coagulation time for the control group was twenty-two minutes.

A group of twenty-six patients with thromboembolic diseases was then studied (Table I). Whereas the mean coagulation time for the control patients was twenty-two minutes, it was only fourteen minutes for this group with intravascular clotting (Fig. 3).

Statistically, this variation between health and disease was significant.* An increased rate of clotting time as determined by this method would, therefore, draw attention to the possibility of intravascular clotting, or confirm it if sus-

*Statistical analysis of the data showed the sigma of the mean to be 0.363 for the control group and 0.587 for the diseased group; the sigma of the difference was 0.690. The critical ratio was found to be 11.6. When Fisher's t was calculated the probability was found to be less than 0.001.

These findings indicate that the data subjected to analysis were of statistical significance.

pected. It would likewise influence both the heparin tolerance curve and the calculated dose of heparin. If the clotting time of a patient is to be maintained with heparin at or above three times his normal or pretreatment level, the desired rate becomes a relative figure rather than one arbitrarily set for all patients.

In a concurrent study, the height and duration of the rise in clotting time following administration of heparin were found by this method to be roughly proportional to the quantity injected, its physical state and medium, and the patient's tolerance for heparin.[4]

TABLE I. CLOTTING TIMES OF PATIENTS WITH THROMBOEMBOLIC DISEASES

CASE NUMBER	DISEASE	COAGULATION TIME (MINUTES)
1	Thrombosis, right femoral artery	11
2	Thrombosis, left popliteal artery	16.5
3	Embolus, left popliteal artery	16
4	Embolus, left popliteal artery	13
5	Embolus, right femoral artery	16
6	Embolus, bifurcation of aorta	14
7	Thrombosis, left popliteal artery	11
8	Thrombosis, right subclavian artery	6.5
9	Thrombosis, left femoral artery	17.5
10	Superior mesenteric artery thrombosis	17
11	Superior mesenteric artery thrombosis	14.5
12	Venous mesenteric thrombosis	13.5
13	Pulmonary infarct	11.5
14	Pulmonary infarcts	14
15	Pulmonary infarct	10
16	Pulmonary infarcts	10
17	Pulmonary infarct	14
18	Bilateral thrombophlebitis, femoral	19.5
19	Superficial and deep femoral thrombophlebitis, left	16
20	Superficial and deep femoral thrombophlebitis, right	13.5
21	Thrombophlebitis, axillary vein (postoperative)	15.5
22	Phlebothrombosis, lower extremity	17.5
23	Thrombophlebitis, lower extremity	13.5
24	Lateral sinus thrombosis	14.5
25	Priapism	17
26	Gangrene, right foot, arterial thrombosis	9
Mean		14

DISCUSSION

Quick,[3] in referring to coagulation time methods, has stated that "trustworthy results can be obtained only by performing the test under constant and rigidly controlled conditions and by excluding all outside agents that influence the coagulation reaction." He has also mentioned the need for a simple, uniform procedure. In discussing the Lee-White test, he said: "This procedure . . . yields as much and as accurate information as any test of coagulation so far devised, but unfortunately 'the test has not been rigidly standardized and at present there is no strict uniformity in the details of the procedure.'"

Ideally, coagulation time should be determined by withdrawing blood without trauma, allowing it to remain in a clean tube unmolested by air currents or motion until that precise instant when the coagulation end point is about to be reached, then suddenly inverting the tube and confirming the presence of a well-formed clot. This may only be approximated in actual practice. By in-

creasing the number of tubes to five and allowing the last one to remain for a longer time undisturbed by agitation or tilting, it is more nearly approached than when fewer tubes are used and the factor of agitation has a greater effect on shortening the coagulation time. The brief interval frequently observed between the clotting times of the fourth and fifth tubes indicates that the last tube was often ready to clot spontaneously when its turn for agitation came (Fig. 3).

The temperature gradient appears to have little influence on the results of this test, which has more tubes and increased accuracy. When the variation in clotting time between similar samples which are run in a water bath at 37° C. and at room temperature, within the seasonal range of extremes mentioned, was found to be one-half minute or less, the water bath was eliminated. With certain other methods,[1, 4] it has also been found that room temperature does not significantly affect the clotting time.

Consistent results on similar samples of blood were obtained by this method by different personnel under different conditions. Such slight variations as occurred represented smaller percentile errors than those usually found with other methods. If a method is accurate to only one minute, when a one-tube procedure is used wherein the tube is gently tilted every thirty seconds to produce a normal clotting range of five to eight minutes, an error of one minute equals 12.5 to 20 per cent. Using three tubes, with a range between nine and fourteen minutes, a one-minute error equals 7 to 11 per cent. With the described five-tube method, however, having a range between eighteen and twenty-six minutes, an error of one minute decreases the percentile difference to between four and five. Certainly, if a shortened coagulation time is to be considered a possible indication of intravascular clotting, a method with 5 per cent error or less is preferable to one with 10 to 20 per cent.

Many clinicians have preferred Dicumarol (3,3′-methylenebis [4-hydroxycoumarin]) to heparin in anticoagulant therapy because the method of determining prothrombin times has been well standardized, whereas methods of coagulation time determinations have varied widely when performed by different personnel in different hospitals. Furthermore, because adverse results can also occur during heparin therapy, it is important to have a method which is consistent and from which as many variables as possible have been removed. Altered coagulation times as determined by the method described here provide an accurate laboratory control in the use of heparin.

SUMMARY

A method for determining the coagulation time of blood is presented. The essentials are atraumatic transfer of blood from vein to receptacle, the use of clean, dry, unscarred glass tubes of uniform size, and minimal agitation or exposure of the final tube.

The method is simple, effective, and as useful clinically as it is in the experimental laboratory. Because it requires only readily available apparatus and eliminates the need for constant temperature, it can be used at the bedside, in the home, office, or hospital with comparable results. It gives repro-

ducible results in the hands of both well-trained and relatively inexperienced personnel, and has been found to give checks of within one minute when performed by different technicians from day to day.

It has produced uniform, accurate results of statistical significance in a group of 150 hospital patients without evidence of intravascular clotting and in a second group of twenty-six patients with thrombosis or embolism, in whom a significant lowering of coagulation time was observed.

It is emphasized that valid and comparable results can be obtained only if a uniform method is used with strict adherence to the selected technique.

We wish to express our appreciation to Mary Elizabeth Shannon, B.S., for her technical assistance in this study.

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NUTRITION FOLLOWING SUBTOTAL GASTRECTOMY OF FOUR TYPES (BILLROTH I AND II, SEGMENTAL, AND TUBULAR RESECTIONS)

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ABNORMAL weight loss after subtotal gastrectomy is a frequent occurrence. Ivy, Grossman, and Bachrach[1] collected 864 cases from the literature, in 42 per cent of which the patients had lost weight following subtotal gastrectomy. In a recent report from this hospital, the average weight after operation of 893 patients who had undergone subtotal gastrectomy was 15.3 pounds below the usual preoperative weight.[2] Many of these patients are asymptomatic except for an inability to gain weight.

This investigation has been conducted to determine the etiological factors of importance causing weight loss after gastric resection so that therapeutic measures may be more intelligently applied. The relative importance of the following factors have been assessed: abnormal fat and nitrogen loss in the stool, decreased oral intake, pancreatic function as assessed by the secretin test following subtotal gastrectomy, and the possible significance of gastrojejunal anastomosis versus gastroduodenal anastomosis with and without preservation of vagus nerve continuity.

Many opinions in the literature concerning postgastrectomy nutrition are clinical impressions. The only reliable methods of investigation presently available to obtain objective information on this subject are metabolic (intake-output balance) studies and accurate records of weight change after operation.

METHODS

This investigation was conducted on thirty-five patients who had undergone the following operations three months to five years previously: Billroth I and Billroth II subtotal gastrectomy, segmental gastric resection,[3] and tubular gastric resection[4, 22, 23] Fig. 1). The patients chosen for investigation were not specially selected as being nutritional problems, nor as having particularly good results. The decision of who should be admitted to the hospital for study rested entirely with the patients.

All patients were admitted to the hospital for a period of eight to ten days, during which time the following studies were performed:

Supported by funds made available to the investigators through institutional grants from the American Cancer Society and the Damon Runyon Fund.

Received for publication, Sept. 26, 1953.

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1. Routine history and physical examination with special inquiry into eating habits and gastrointestinal symptoms.

2. Determination of hemoglobin, erythrocytes, leukocytes, and differential, mean cell diameter, reticulocytes, blood urea nitrogen, and total serum protein.

3. Roentgenologic examination with barium of the esophagus and upper gastrointestinal tract, as well as estimation of the transit time to the cecum in most cases.

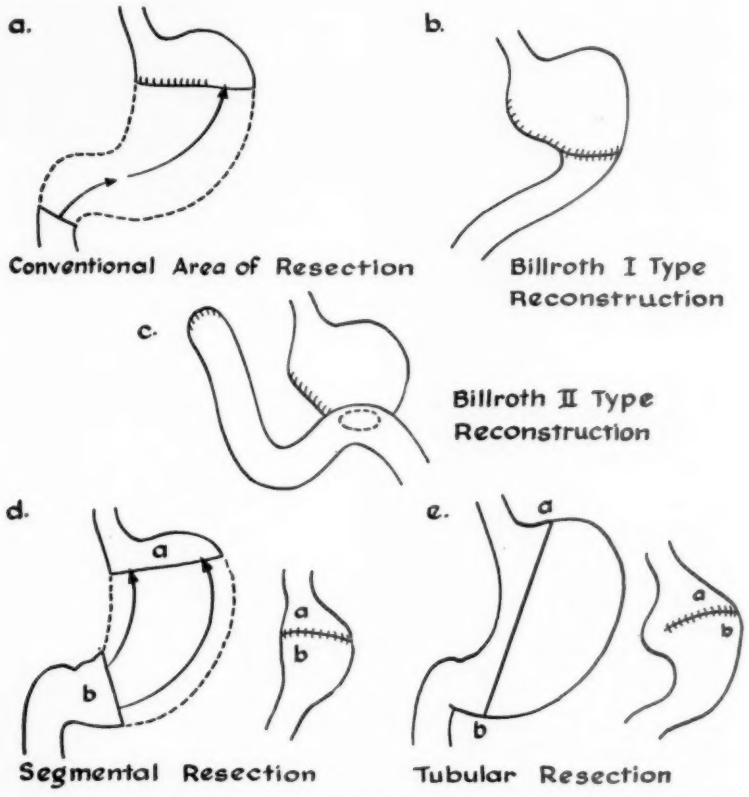

Fig. 1.—*a*, The conventional area of gastric resection in the treatment of peptic ulcer is the distal three-quarters of the stomach.

b, Billroth I reconstruction is an end-to-end anastomosis between the duodenum and cut end of the stomach with a Hofmeister closure of the lesser curvature.

c, Billroth II reconstruction with gastrojejunostomy and short afferent loop.

d, Segmental gastric resection is a three-quarter resection principally of the acid-producing area. Anastomosis is performed between the antrum and a very small fundic pouch. A Heineke-Mikulocz pyloroplasty is mandatory to permit satisfactory gastric emptying.

e, Tubular gastric resection is an excision of two-thirds of the stomach. The area of excision is the entire length of the greater curvature. Reconstruction is by means of transverse gastroplasty without interruption of the vagus nerves. The antrum remains. A pylorotomy is optional, and is performed only for obstruction and in the presence of massive hemorrhage for a duodenal ulcer. Duodenal ulcer is the chief indication for tubular resection.

4. Measurement of fecal fat and nitrogen excretion. Three- to four-day stool collections were used, with carmine dye orally to mark the interval. A weighed diet containing 145 Gm. of carbohydrate, 80 Gm. of fat, and 80 Gm. of protein with a total of 1,620 calories was offered, and the amount eaten was calculated. Intake was charted as for a person with diabetes, so that the actual quantity ingested was known. Fecal fat was determined by the method of Fowweather,[8]

and fecal nitrogen by the method of Kjeldahl.[9] Fat excretion in the stool was reported as percentage of ingested fat. Fecal nitrogen was reported in grams per day in the stool.

5. Study of pancreatic function by aspiration of the intestinal content below the gastric pouch before and after administration of secretin intravenously. Two tubes were placed in the gastrointestinal tract under fluoroscopy. One tube with a mercury balloon at the tip was placed as close as possible to the ampulla of Vater. A nine-hole gastric tube was positioned in the gastric pouch. Both tubes were placed on suction. The intestinal aspirate was collected under ice and frozen, and the following determinations were made: volume, pH, amylase, lipase, trypsin, and bicarbonate. Two thirty-minute fasting samples were collected, following which secretin, 1 unit per kilogram, was administered intravenously, and three twenty-minute samples were collected. The division of samples after secretin stimulation serves no useful purpose; therefore, all results are combined for the one-hour collection period. The pH was determined with a glass electrode. Amylase was determined by the method of Somogyi,[10] trypsin by the method of Dorothy Anderson,[11] and lipase by a modification of the method of Cherry and Crandall.[12]

RESULTS

Metabolic Studies Following Billroth II Procedure.—Nine patients who had undergone Billroth II resection were studied by intake-output balance methods. The weight loss, fat and nitrogen excretions in the stool, and estimated percentage of normal food intake since operation are tabulated in Table I. The normal fat excretion on a diet containing 80 Gm. of fat is 5 per cent of intake.[5] Over 10 per cent fecal excretion of fat intake is distinctly abnormal; 7.5 to 10 per cent is a borderline abnormality. On this basis, three of the nine patients were excreting abnormally large quantities of fat in the stool, one was borderline, and five were within normal limits. One patient, J. M., was excreting 37 per cent of ingested fat in the stool—the greatest loss encountered in this study. Weight loss was not entirely due to fat loss since two patients with normal fat excretion lost twenty-five and eighteen pounds, respectively. These patients were eating only 60 to 75 per cent of the amount ingested prior to surgery.

After the Billroth II procedure, three patients were excreting an excessive amount of nitrogen in the stool. The upper limit of normal excretion is 2 Gm. of nitrogen per day when the diet does not contain an excess of indigestible nitrogenous or nonnitrogenous roughage.[6] Diarrhea was prominent in only two of the patients; one of these patients was excreting an abnormal amount of both fat and nitrogen.

Metabolic Studies Following Billroth I Procedure.—Following Billroth I procedure, five patients underwent investigation as outlined (Table II). Four of the five lost an excessive amount of weight. Only one of the five patients excreted an abnormal amount of fat (10 per cent of intake). Nitrogen excretion was normal in all cases. Weight loss in all instances was closely related to decreased oral intake.

TABLE I. FECAL FAT AND NITROGEN LOSS IN RELATION TO WEIGHT CHANGE AFTER BILLROTH II GASTRIC RESECTION

PATIENT	AGE (YR.)	WEIGHT LOSS (POUNDS)	INTERVAL BETWEEN OPERATION AND STUDY (MO.)	FAT LOSS* AS PER CENT OF INTAKE	NITROGEN† LOSS IN STOOL (GRAMS PER DAY)	PER CENT OF NORMAL FOOD INTAKE	SMALL BOWEL TRANSIT TIME	GASTRIC EMPTYING TIME (MIN.)	DIAGNOSIS
1. H. E.	66	10	16	4.4	0.53	75+	2 hr.+	10	Duodenal ulcer
2. R. C.	59	15	10	9.0	1.27	75+	2 hr.+	10	Duodenal ulcer
3. T. W.	40	18	24	3.4	0.50	60	6 hr.	Immediate	Carcinoma of stomach
4. A. A.	68	5	36	6.8	1.30	100	5 hr.	15	Carcinoma of stomach
5. O. G.	56	7	42	17.7	2.45	100	2 hr.	—	Carcinoma of stomach
6. J. M.	67	30	26	37	6.67	75	20 min.	5	Gastric ulcer
7. W. W.	66	0	82	3	0.31	100	2 hr.+	60	Duodenal ulcer
8. A. R.	51	25	14	4	0.67	75	30 min.	15	Gastric ulcer
9. J. F.	56	25	24	10	2.30	50-75	—	30	Gastric ulcer

*Normal fat loss in stool when patient is ingesting 80 Gm. of fat in diet is 5 to 7.5 Gm. (7.5 to 10 Gm. is borderline, over 10 Gm. is abnormal).

†Normal fecal nitrogen excretion is under 2 Gm.

TABLE II. FECAL FAT AND NITROGEN LOSS IN RELATION TO WEIGHT CHANGE AFTER BILLROTH I GASTRIC RESECTION

PATIENT	AGE (YR.)	DIAGNOSIS	WEIGHT CHANGES (POUNDS)	INTERVAL BETWEEN OPERATION AND STUDY (MO.)	FAT LOSS IN PER CENT OF INTAKE	NITROGEN LOSS (GRAMS PER DAY)	PER CENT OF NORMAL FOOD INTAKE	SMALL BOWEL TRANSIT TIME	GASTRIC EMPTYING TIME (MIN.)
1. D. E.	24	Duodenal ulcer	−18	7	4.5	0.66	50	30 min.	10
2. A. A.	77	Gastric ulcer	−15	18	6	1.68	75	7 hr.	20
3. I. M.	70	Jejunal ulcer	−20	18	7	0.5	50	3.5 hr.	10
4. C. H.	67	Gastric ulcer	−20	8	5	1.06	75	2 hr.+	20
5. A. K.	65	Duodenal ulcer	−25	6	10	1.2	50	6 hr.	30

TABLE III. FECAL FAT AND NITROGEN LOSS IN RELATION TO WEIGHT CHANGE AFTER SEGMENTAL GASTRIC RESECTION

PATIENT	AGE (YR.)	DIAGNOSIS	WEIGHT CHANGE (POUNDS)	INTERVAL BETWEEN OPERATION AND STUDY (MO.)	FAT LOSS IN PER CENT OF INTAKE	NITROGEN LOSS (GM./DAY)	PER CENT OF NORMAL FOOD INTAKE	SMALL BOWEL TRANSIT TIME	GASTRIC EMPTYING TIME (MIN.)
1. F. B.	61	Duodenal ulcer	−28	12	6	—	75	3 hr.	120
2. E. A.	52	Duodenal ulcer	−42	9	7.3	1.21	50	2 hr.	10
3. O. L.	52	Duodenal ulcer	−80	17	12	2.2	50	40 min.	40
4. H. M.	36	Duodenal ulcer	−30	12	11.6	1.4	60	2 hr.+	5
5. A. L.	65	Duodenal ulcer	−30	14	10.5	0.68	75	—	30
6. C. S.	69	Duodenal ulcer	−15	13	9.2	0.95	75	3 hr.	20
7. F. O.	53	Duodenal ulcer	−20	17	5.8	1.2	75	4 hr.	40
8. E. M.	50	Duodenal ulcer	+16	8	5.6	1.58	100	1.5 hr.	90
9. A. R.	50	Duodenal ulcer	−20	24	3.5	1.2	50	2 hr.	30
10. E. G.	63	Duodenal ulcer	−10	17	7	1.2	60	6 hr.	40
11. J. W.	60	Duodenal ulcer	−20	16	9	1.1	100	—	30
12. W. O.	51	Duodenal ulcer	−35	13	2.1	—	50	2 hr.+	45
13. L. L.	64	Duodenal ulcer	−15	16	5	0.75	75	4.5 hr.	90
14. J. P.	49	Duodenal ulcer	−30	26	12	0.98	60	4 hr.	10
15. F. R.	58	Duodenal ulcer	−50	23	5	0.90	60	9 hr.+	60
16. F. E.	46	Duodenal ulcer	−18	18	11	1.4	60	4 hr.	30

TABLE IV. FECAL FAT AND NITROGEN LOSS IN RELATION TO WEIGHT CHANGE AFTER TUBULAR GASTRIC RESECTION

PATIENT	AGE (YR.)	WEIGHT LOSS (POUNDS)	INTERVAL BETWEEN OPERATION AND STUDY (MO.)	FAT LOSS IN PER CENT OF INTAKE	NITROGEN LOSS IN STOOL (GRAMS PER DAY)	PER CENT OF NORMAL FOOD INTAKE	SMALL BOWEL TRANSIT TIME	GASTRIC EMPTYING TIME (MIN.)	DIAGNOSIS
1. W. H.	44	20	4	4	1.36	75	—	—	Duodenal ulcer
2. L. L.	40	20	5	6	2.6	75	2 hr.+	30	Duodenal ulcer
3. G. Cl.	39	25	6	4.5	0.4	50-60	—	—	Duodenal ulcer
4. G. C.	44	30	6	5	0.5	50-60	30 min.	30	Duodenal ulcer
5. Y. R.	46	10	5	4.3	1.3	90	—	40	Duodenal ulcer

Metabolic Studies Following Segmental Resection.—Following segmental gastric resection,[3] sixteen patients underwent intake-output balance tests (Table III). Five were excreting distinctly abnormal quantities of fat in the stool, three were borderline, and eight patients were excreting normal quantities of fat in the stool.

Only one patient excreted a slightly elevated quantity of nitrogen in the stool.

Metabolic Studies Following Tubular Resection.—Five patients, four of whom had lost an excessive amount of weight following tubular gastric resection,[4, 22, 23] were investigated (Table IV). No abnormal or borderline abnormality of fat excretion exists. One patient excreted a slightly elevated quantity of nitrogen in the stool. Diarrhea is not present, but decreased oral intake is prevalent in this group of five patients.

Results of Metabolic Studies.—On the basis that 5 to 7.5 per cent of fat intake excreted in the stool is normal, 7.5 to 10 per cent is borderline, and over 10 per cent is distinctly abnormal, thirty-five patients who had undergone subtotal gastrectomy were studied. Eight of the thirty-five patients were excreting abnormal quantities of fecal fat (up to 37 per cent of oral intake in one patient). Five of the thirty-five patients were excreting borderline quantities of fecal fat, and the remaining twenty-two patients were within normal limits.

A distinctly abnormal loss of nitrogen in the stool (6.67 Gm. per day) was found in only one patient.

On the basis of the patients' opinion of the quantity of food ingested compared to the amount ingested preoperatively, when well, one can state that weight loss following gastrectomy is often due to decreased caloric intake. All patients were repeatedly questioned on oral intake and closely observed while in the hospital in order to obtain a reliable approximation of oral intake. While this method is subject to error in individual cases, we feel it supplies excellent information on groups such as this. Of 172 cases of tubular and segmental gastric resection, all patients who weighed the same as before operation (within five pounds of their usual weight) volunteered they were eating 100 per cent of what they did before operation. Many patients who had lost an excessive amount of weight (over twenty pounds) either could not ingest the 1,620 calorie diet while in the hospital or felt uncomfortably full after doing so. The dumping syndrome or early postprandial distress was precipitated by single large meals, which was a limiting factor in some cases, preventing a larger total caloric intake. These patients had learned that they could avoid the distress of palpitation, perspiration, nausea, drowsiness, and weakness by eating smaller quantities of food. Smaller meals at frequent intervals only partially compensates for the decreased caloric intake in these patients. Diets with the caloric content composed of variable amounts of fat and carbohydrate are being investigated at the present time.

Pancreatic Function Following Subtotal Gastrectomy.—In addition to intake-output balance study of fat and nitrogen excretion, secretin tests were performed on twenty-eight of the previously mentioned patients and on five

normal persons, as outlined under Methods. It is not unreasonable to assume that pancreatic secretion would be reduced after subtotal gastrectomy. The effective stimuli to pancreatic secretion are (1) vagus nerve stimulation, (2) secretin mechanism—lowering of the pH of the duodenum to 4 leads to the production of secretin, which in turn stimulates external pancreatic and biliary secretion.

Following subtotal gastrectomy the acid-producing capacity of the stomach is markedly lowered and the vagus nerve supply to the pancreas is probably interrupted. Two patent sources of pancreatic stimulation are thereby lost or diminished in effect.

Since patients with steatorrhea on a pancreatic basis with excessive fat loss in the stool do show a marked depression of response to secretin, this test was used to evaluate pancreatic function following subtotal gastrectomy.

Results of Pancreatic Function Tests.—A group of five young normal persons was given the secretin test as outlined. The results are illustrated in Table V. The tests performed on patients who had undergone subtotal gastrectomy are illustrated in Tables VI, VII, VIII, and IX.

TABLE V. SECRETIN TEST ON NORMAL HUMAN BEINGS

PATIENT	VOLUME (C.C.)	pH	HCO_3 (C.C.)	AMYLASE (SOMOGYI UNITS)	TRYPSIN (GELATIN TUBES LIQUEFIED)	LIPASE (C.C. OF 0.05 N NaOH*)
1. F. S.	222	7-9	155	45,600	2-9	0.55
2. P. L.	110	8	63	16,550	6-9	—
3. M. E.	31.5	7-8	81	17,450	3-9	0.50
4. K. S.	73	7-8	28	15,200	4-9	0.38
5. B. J.	93	7+	30	46,400	4-6	0.90

Dilutions of duodenal juice used:

Tube		*Duodenal Juice*
1	0.125	1-100
2	0.25	1-100
3	0.5	1-100
4	1.0	1-100
5	0.2	1-10
6	0.4	1-10
7	0.8	1-10
8	1.6	1-10
9	0.32	undiluted
10		

*The lowest dilution in which the gelatin has liquefied in one or both tubes (duplicates) is considered as the reading.

All pancreatic function tests performed on the Billroth I group of patients are completely within normal limits in all respects. One patient, J. M., showed no pancreatic response to secretin stimulation following the Billroth II procedure. This patient has severe diarrhea with excessive fat excretion in the stool (37 per cent of intake), which has been partially relieved by pancreatin in large doses. A corrective diet for sprue and Banthine were ineffective in the treatment of this patient. This was the only patient examined in whom reduced pancreatic function probably is of importance in increased fat loss in the stool following subtotal gastrectomy. Following Billroth II gastrectomy, two other patients, H. E. and T. W., had below average volume responses to secretin; however, fat excretion is well within normal limits and nitrogen loss in the stool in the patients is completely normal.

TABLE VI. RESULTS OF SECRETIN TEST FOLLOWING BILLROTH II SUBTOTAL GASTRIC RESECTION

PATIENT	VOLUME (C.C.)	pH	HCO_3	AMYLASE (SOMOGYI UNITS)	TRYPSIN (GELATIN TUBES LIQUEFIED)	LIPASE (C.C. OF 0.05 N NaOH)	FAT EXCRETION (PER CENT OF INTAKE)	WEIGHT CHANGE (POUNDS)	PANCREATIC FUNCTION
1. H. E.	3.5	7	0	133,750	qns	qns	4.4	−10	Abnormal
2. B. C.	100	7+	109.1	111,600	5.6	1.9	9	−15	Normal
3. T. W.	14	7.3	0	31,100	4-9	0.72	3.4	−18	Abnormal
4. J. M.	0.5	8	—	—	—	—	37	−30	Abnormal
5. J. F.	43	7+	35	173,700	—	—	10	−25	Normal
6. A. S.	208	7+	6.2	56,250	7-9	0.2	4	−25	Normal

TABLE VII. RESULTS OF SECRETIN TEST FOLLOWING BILLROTH I SUBTOTAL GASTRIC RESECTION

PATIENT	VOLUME (C.C.)	pH	HCO_3	AMYLASE (SOMOGYI UNITS)	TRYPSIN (GELATIN TUBES LIQUEFIED)	LIPASE (C.C. OF 0.05 N NaOH)	FAT EXCRETION (PER CENT OF INTAKE)	WEIGHT CHANGE (POUNDS)	PANCREATIC FUNCTION
1. D. E.	235	7-8	58	140,000	5-7	0.6	4.5	−26	Normal
2. A. A.	84	7+	24	111,600	5-7	1.0	6	−15	Normal
3. I. M.	172	6.5	68	100,500	5-6	0.5	7	−10	Normal
4. C. H.	57	8+	134	130,500	5-7	1.3	5	−25	Normal
5. A. K.	33	7.5	85	63,200	4-5	0.09	10	−25	Normal

TABLE VIII. RESULTS OF SECRETIN TEST FOLLOWING SEGMENTAL GASTRIC RESECTION

PATIENT	VOLUME (C.C.)	pH	HCO_3	AMYLASE (SOMOGYI UNITS)	TRYPSIN (GELATIN TUBES LIQUEFIED)	LIPASE (C.C. OF 0.05 N NaOH)	FAT EXCRETION (PER CENT OF INTAKE)	WEIGHT CHANGE (POUNDS)	PANCREATIC FUNCTION
1. J. P.	8.2	7-8	0	3,840	7		12	–40	Abnormal
2. F. E.	72.5	7-7.5	22	70,450	4-7	3.3	11	–24	Normal
3. A. R.	76	8.5-9.3	154.1	98,400	—	9.9	3.5	–20	Normal
4. E. G.	24.5	8.1-8.5	69.0	78,050	—	2.0	7	–10	Normal
5. J. W.	220	7.6-7.9	55	73,500	4-6	0.5	9	–20	Normal
6. C. A.	63	8.9-9.1	96.3	22,740	7-8	0.04	—	–15	Normal
7. E. A.	60	7.4-7.6	13.6	3,650	4-9	13.4	7.3	–22	Normal
8. E. M.	44	7.0	59	4,100	3-9	0.45	5.6	+16	Normal
9. O. L.	21.9	8-8.4	92	45,000	6	3.9	12	–80	Abnormal
10. H. M.	199	8.8-8.6	52	51,400	6-7	9.5	11.6	–30	Normal
11. A. L.	110	7.3-8.7	54.2	54,040	6-7	0.4	10.5	–50	Normal
12. C. S.	72	7.8-7.9	149	47,400	7-8	0.2	9.2	–20	Normal
13. F. D.	11.2	8.2	97	42,350	—	2.0	5.8	–20	Abnormal

TABLE IX. RESULTS OF SECRETIN TEST FOLLOWING TUBULAR GASTRIC RESECTION

PATIENT	VOLUME (C.C.)	pH	HCO_3	AMYLASE (SOMOGYI UNITS)	TRYPSIN (GELATIN TUBES LIQUEFIED)	LIPASE (C.C. OF 0.05 N NaOH)	FAT EXCRETION (PER CENT OF INTAKE)	WEIGHT CHANGE (POUNDS)	PANCREATIC FUNCTION
1. W. H.	47.7	7-8	50.1	318,700	6	2.3	4	–20	Normal
2. L. L.	0.9	+8		441,600			6	–30	Abnormal
3. G. C.	56.1	7.6-9.6	0	157,200	8	0.5	5	–30	Normal
4. Y. R.	124	+7	66	328,000	4	0.1	4.3	–10	Normal

Of sixteen patients who were examined following segmental resection, two (J. P. and O. L.) had excessive fat loss and showed a subnormal pancreatic response to secretin (Table VIII). Neither patient was excreting excessive quantities of nitrogen in the stool.

Following tubular gastric resection, one patient, L. L., had a subnormal response to secretin; however, this patient's excretion of fat in the stool was within the normal range.

Summary.—The pancreatic function of twenty-eight patients following subtotal gastrectomy of four types was examined by means of the secretin test. Eleven of these patients were excreting distinctly abnormal or borderline quantities of fecal fat. In only three of the eleven patients could excessive fat loss possibly be attributed to pancreatic insufficiency on the basis of the secretin test. In only one of these patients was there an excessive excretion of nitrogen in the stool, characteristic of pancreatic insufficiency.

DISCUSSION

The possible causal factors of weight loss following gastrectomy have been stated by many authors[6, 16-18] to be one or more of the following: increased fat loss in the stool due to diminished pancreatic flow or imperfect mixing of foods and ferments; increased intestinal motility; loss of gastric digestion; and loss of gastric reservoir.

For the purposes of this investigation, normal weight is the usual weight of the patient for five years prior to operation, so that abnormal changes due to diet or obstruction immediately preceding the operation are eliminated. On this basis, thirty-two of the thirty-five patients lost weight. The average loss was twenty-one pounds.

Gastroduodenal continuity does not prevent excessive fat loss in the stool, since this occurred following segmental gastric resection.

Increased fat loss is not constantly accompanied by diarrhea or increased small bowel motility on roentgen examination. One patient who excreted 37 per cent of fat intake had a twenty-minute small bowel transit time, while a patient, following the same operation, who had a thirty-minute small bowel transit time was excreting only 4 per cent of fat intake (Table I). A thin barium meal was used in all cases. A mixture of barium and food possibly would give a more accurate picture of small bowel motility after partial gastric resection. Many patients, after partial gastrectomy, experience abdominal cramps after eating, which last for ten to thirty minutes and are aggravated by further eating. This probably represents hypermotility of the upper small bowel and may be important in separating the ingested food from pancreatic enzymes.

The gastric emptying time following Billroth II resection is rapid, usually being under thirty minutes (Table I). This circumstance is corroborated by Dr. Rigler and Dr. Nice of the Radiology Department of the University of Minnesota, who have examined a much larger series than is here reported.[21] Gastric emptying time after tubular and segmental gastric resection is usually thirty minutes or longer (Tables III and IV). No correlation exists, however, between rapid gastric emptying and weight loss or increased fecal fat excretion.

Increased fecal fat excretion occurs frequently following segmental resection and Billroth II subtotal gastric resection. With one exception, however, the fat losses were not great and cannot account entirely for major weight losses in patients. Excessive fat loss has not occurred after tubular gastric resection where vagus nerve continuity is preserved. The possibility exists that vagus nerve continuity is of importance in preventing excessive fecal fat excretion.

That these effects of vagotomy are not due to pancreatic insufficiency is suggested by the predominantly normal secretin tests and a normal stool nitrogen in all but one patient in this study. Furthermore, Routley and co-workers,[14] working with dogs, found that vagotomy had no effect on pancreatic functions when secretion was stimulated by food, acid, and alcohol in the upper gastrointestinal tract.

Fox and Grimson[13] studied nine patients who had lost weight following vagotomy. Three of the patients had undergone vagotomy alone, six vagotomy and gastroenterostomy. Eight of the nine patients were excreting an excessive amount of fat. One patient was excreting 33 per cent of intake (normal is 5 per cent).

Vagotomy may adversely affect intestinal enzyme secretion. Savich and Soshestvensky[15] have demonstrated an increased secretion of succus entericus in the cat on vagal stimulation both in volume and enzyme concentration. A large dose of atropine diminished or even completely arrested the intestinal secretion of these enzymes. This problem deserves further investigation.

Of the factors assessed to account for weight loss in the thirty-five patients who had undergone partial gastrectomy, decreased oral intake or loss of gastric reservoir is of greatest importance. Many patients whose fecal fat and nitrogen excretion were within normal limits had lost over ten pounds of weight. These patients were eating considerably less in twenty-four hours, however, than prior to surgery. A disagreeable feeling of fullness or precipitation of the dumping syndrome or rapid satiety prevented many of these individuals from ingesting greater quantities even with six meals a day.

Increased fecal fat excretion is an additional cause of weight loss in some patients. Excessive fat excretion may be caused by improper mixing of food and pancreatic enzymes due to rapid upper small bowel motility or due to the effects of intestinal vagotomy in some cases. Pancreatic insufficiency as a cause of weight loss after partial gastrectomy is not tenable for the following reasons: (1) normal secretin tests were obtained in the vast majority of cases tested; (2) pancreatin is of little or no value in the treatment of abnormal fat loss in the stool after gastrectomy;[16] (3) normal stool nitrogen excretion was uniformly found; (4) weight loss occurs regularly in the absence of either excessive fat or nitrogen loss in the stool.

The weight loss in this group of patients is high when postoperative weight is compared to the usual preoperative weight. The weight loss below ideal weights,[19, 20] as calculated for height and body build and considered in terms of longevity, is considerably less in most cases. A comparison of usual weight, present weight, and ideal weight for all cases is illustrated in Table X. In many instances, the postoperative weight loss has brought patients from an overweight state to within ideal weight range.

TABLE X. WEIGHT CHANGE IN ALL PATIENTS

NAME	TYPE OF OPERATION	USUAL PREOPERATIVE WEIGHT	PRESENT POSTOPERATIVE WEIGHT	IDEAL WEIGHT[19, 20]
1. J. W.	Segmental	170	150	145-156
2. E. G.	Segmental	135	125	136-147
3. E. A.	Segmental	180	138	153-164
4. E. M.	Segmental	130	146	153-164
5. L. L.	Segmental	145	130	137-147
6. O. L.	Segmental	250	170	157-168
7. F. E.	Segmental	138	120	133-143
8. F. O.	Segmental	175	155	157-168
9. F. R.	Segmental	200	150	157-168
10. C. S.	Segmental	165	150	153-164
11. F. B.	Segmental	178	150	153-164
12. H. M.	Segmental	185	155	153-164
13. R. C.	Billroth II	160	145	134-144
14. D. E.	Billroth I	118	100	107-115
15. O. G.	Billroth II	175	168	161-173
16. A. And.	Billroth I	130	115	145-156
17. C. H.	Billroth I	135	115	137-147
18. A. A.	Billroth II	145	140	134-144
19. T. W.	Billroth II	148	130	141-151
20. J. M.	Billroth II	140	110	145-156
21. A. K.	Billroth I	110	85	110-118
22. I. M.	Billroth I	175	155	145-156
23. J. F.	Billroth II	150	125	141-151
24. H. E.	Billroth II	155	145	117-125
25. W. O.	Segmental	190	155	157-168
26. A. S.	Billroth II	130	105	127-135
27. J. P.	Segmental	180	150	176-189
28. W. W.	Billroth II	220	220	166-178
29. A. L.	Segmental	160	130	124-132
30. W. H.	Tubular	160	140	156-164
31. Y. R.	Tubular	160	150	156-164
32. L. Lig.	Tubular	160	140	141-151
33. G. Cl.	Tubular	170	145	130-140
34. G. C.	Tubular	165	135	137-149
35. A. R.	Billroth II	150	125	124-132

TABLE XI. SUMMARY OF WEIGHT, FECAL FAT, FECAL NITROGEN LOSS, AND AVERAGE ORAL INTAKE

TYPE OF OPERATION	AVERAGE WEIGHT LOSS (POUNDS)	AVERAGE* PER CENT OF WEIGHT LOSS	AVERAGE FECAL FAT LOSS (GRAMS)	AVERAGE FECAL NITROGEN LOSS (GRAMS)	AVERAGE ORAL INTAKE PER CENT OF PREOPERATIVE INTAKE
Billroth I	19.6	16.2	6.56	1.05	65
Billroth II	15	10.3	9.50	1.79	80
Segmental	28.7	15	7.66	1.04	67
Tubular	21	12.8	4.76	1.23	66

*Per cent of weight loss is calculated as follows: $\frac{\text{Weight loss}}{\text{Usual preoperative weight}} \times 100$ and allows comparison of weight loss in patients of all weights.

A summary of average weight loss, average percentage of weight loss, average fat loss in the stool, average nitrogen loss, and average estimated oral intake is given in Table XI. The smaller average weight loss after the Billroth II operation is probably due to the fact that these patients had been operated upon many years prior to the study and had had opportunity to adjust to the altered gastrointestinal continuity. These patients could eat more, 80 per cent of the previous amount, than patients operated upon recently—segmental and tubular gastric resection patients.[4, 22, 23]

The hemogram made on all patients was normal in each instance, with one exception—a hypochromic, iron-deficiency anemia which occurred in a female patient following a Billroth II type gastric resection. A hemoglobin under 12 Gm. was considered abnormal.

From these studies, it would appear that a more effective therapeutic accomplishment is to be achieved by directing the attention and the interest of the patient to the goal of an increased caloric intake following subtotal gastrectomy, rather than toward efforts directed at increasing fat absorption from the gastrointestinal tract.

SUMMARY AND CONCLUSIONS

1. Thirty-five patients with subtotal gastrectomy were studied from three months to five years following operation to determine the etiological factors of importance causing weight loss.

2. Fat and nitrogen excretion in the stool was determined while the patients were hospitalized on a weighed intake of fat 80 Gm., protein 80 Gm., and carbohydrate 145 Gm. Three of nine patients excreted an abnormally large amount of fecal fat after Billroth II gastrectomy. One of five patients excreted a borderline quantity of fat after the Billroth I procedure. Segmental resection resulted in an abnormally large fat loss in the stool in five of sixteen patients. Fat excretion was normal in the five patients who underwent tubular resection.[4, 22, 23]

3. Excessive stool nitrogen loss is an infrequent occurrence following subtotal gastrectomy.

4. Thirty-two of the thirty-five patients who were studied lost weight. The average loss was twenty-one pounds. Large weight losses are possible with normal fat and nitrogen excretion in the stool and are closely related to decreased food intake despite many meals per day. Weight loss is considerably less when compared to ideal weights for each individual concerned.

5. Large weight loss is not closely related to rapid small bowel transit time or diarrhea.

6. Gastroduodenal continuity does not prevent excessive fat loss in the stool, since this occurred following segmental gastric resection.

7. Pancreatic function, as assessed by the secretin test, following subtotal gastric resection is completely normal in most patients. The possibility exists that rapid small bowel transit separates the food and enzymes in the presence of normal pancreatic function.

8. Of the factors assessed to account for weight loss in thirty-five patients after subtotal gastrectomy, decreased oral intake or loss of gastric reservoir capacity is of greatest importance. Increased fecal fat excretion is an additional cause of weight loss in some patients.

9. Investigation for the future would be most advantageously directed toward increasing caloric intake in patients following subtotal gastrectomy.

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THE EFFECT OF PERORAL BARIUM IN PARTIAL OBSTRUCTION OF THE SMALL BOWEL

An Experimental Study

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IT HAS been recognized for many years that the oral administration of barium in the presence of a potentially obstructing lesion of the colon is a dangerous practice which may result in complete obstruction. Such occurrences have been reported by Stalker,[5] Orgel,[4] and others, who have emphasized the value of performing a barium enema before the upper gastrointestinal series if there is any indication that the patient may have a lesion of the large bowel. Golob[3] has noted that barium remaining in the normal colon will become a stony, hard impacted mass, if not evacuated in a reasonable length of time, because of the function of the colon in absorption of water, and he has reported cases of complete obstruction developing on this basis. From a rational point of view, it would seem unlikely that the same process would occur in the partially obstructed small bowel since the small bowel is concerned primarily with secretion, digestion, and absorption of nutriments, rather than with fluid absorption. According to Best and Taylor,[1] "As a result of the sensitivity of the mucosa to mechanical stimuli, undigested food residues or other unabsorbable solid material will in their passage through the (small) bowel cause excitation of the glands and so increase the fluidity of the intestinal contents." Nevertheless, quite recently Cantor and his associates[2] condemned the use of the upper gastrointestinal series in the presence of suspected partial obstruction of the small intestine, because of the fear that the obstruction might be made complete, unless a long intestinal tube was first passed to the point of obstruction so that the dilute barium could be recovered promptly by suction, after the site of obstruction was demonstrated radiologically.

The present study was undertaken as a result of experiences with several clinical cases of chronic progressive small bowel obstruction, which in most instances were later proved to be on the basis of small bowel neoplasm. The radiologists concerned advocated the administration of oral barium as a diagnostic measure, and felt that there was no likelihood of the barium producing complete obstruction. However, certain of the clinicians concerned felt that the possibility of the development of complete obstruction was a real one.

METHODS

Unselected mongrel dogs of various sizes were used, and all operative procedures were carried out under aseptic conditions with veterinary Nembu-

Received for publication, Aug. 21, 1953.

tal anesthesia. The animals were kept in a fasting state for twelve hours before each operation, and given only liquids for twenty-four hours after recovery from anesthesia, after which the regular kennel diet was resumed. As the initial procedure in each animal, a double thickness of reactive cellophane about 2 cm. in width was placed circumferentially around the terminal ileum 5 to 20 cm. proximal to the ileocecal valve. The cellophane strip, which was placed snugly against the bowel wall without constricting the bowel, was fixed in place by two nonabsorbable sutures at the ends, which were then buried in mesentery so that a smooth surface presented to the peritoneal cavity. It had been planned to follow the development of obstruction with scout films of the abdomen, but the latter were found to be rather difficult to interpret in the earliest studies, and this method was therefore abandoned in favor of re-exploration, merely to determine the presence and degree of obstruction. The second exploration was carried out in most instances seven to fourteen days after implantation of cellophane, except for two animals observed six and seven weeks, respectively. A short time (one to eleven days) after re-exploration, barium sulfate, in the usual dilution, was given through a Levin tube passed into the stomach. The volume of the barium suspension administered varied between 100 and 150 c.c., depending on the size of the animal. Films of the abdomen were taken twelve or twenty-four hours after barium administration, and when there was marked stasis of barium in the small bowel, the progress of the meal was followed longer, in two cases for five days, with repeated films. Shortly after the last x-ray film was taken, the animal was sacrificed and autopsied, or subjected to laparotomy, at which time the involved portion of the intestine was resected between clamps. In some instances, this was done immediately after the last film was made, so that a direct comparison could be made between the roentgenologic findings and the autopsy material. In other animals, an interval of two to five days elapsed between the last film and inspection of the bowel. The degree of obstruction, the fluidity and character of the intestinal contents, and the size of the lumen of the bowel at the site of obstruction were noted in each instance.

RESULTS

Reactive cellophane was wrapped around the terminal small bowel of 38 dogs. Partial small bowel obstruction developed at the site of the cellophane in 30 animals, but because of the death of 14 animals for various reasons, only 16 animals were carried through the complete series of studies.

The gross picture seen at operation and at autopsy after snug encirclement of the small bowel by cellophane was a remarkably uniform one of chronic partial small bowel obstruction. Characteristically, the cellophane was covered, even one week after its application, by serosa, and surrounded by slightly edematous tissue, showing microscopically a marked inflammatory reaction, as well as fibrosis. In most animals, the lumen of the bowel at the site of obstruction was 3 to 6 mm. in diameter (Fig. 1). No mucosal ulceration was seen in any case. The bowel proximal to the obstructing lesion was dilated to two to three times normal size with thick hypertrophic walls, but distal to the site of obstruction, the bowel was collapsed slightly (Fig. 2). The

proximal dilatation usually involved 30 to 40 cm. of small bowel, although in a few cases much more of the bowel was involved. Although regular observations in the kennels were not made, in so far as could be determined, the dogs continued to take food and to have bowel movements, and vomiting was a rare occurrence. This preparation was an effective means of producing a high grade of partial small bowel obstruction fairly consistently.

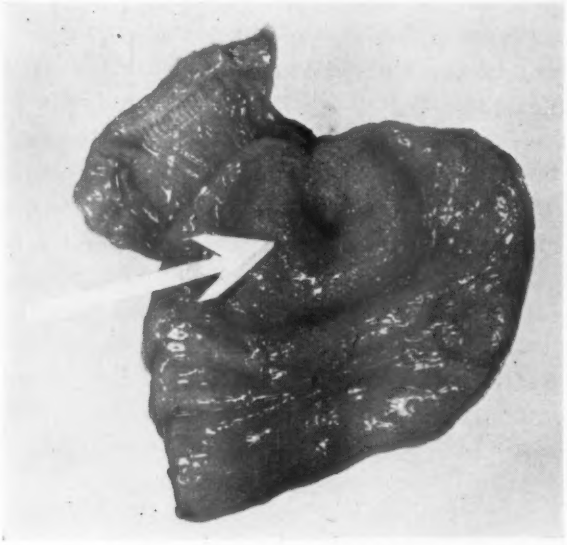

Fig. 1.—Open bowel showing diameter of the lumen at the site of obstruction.

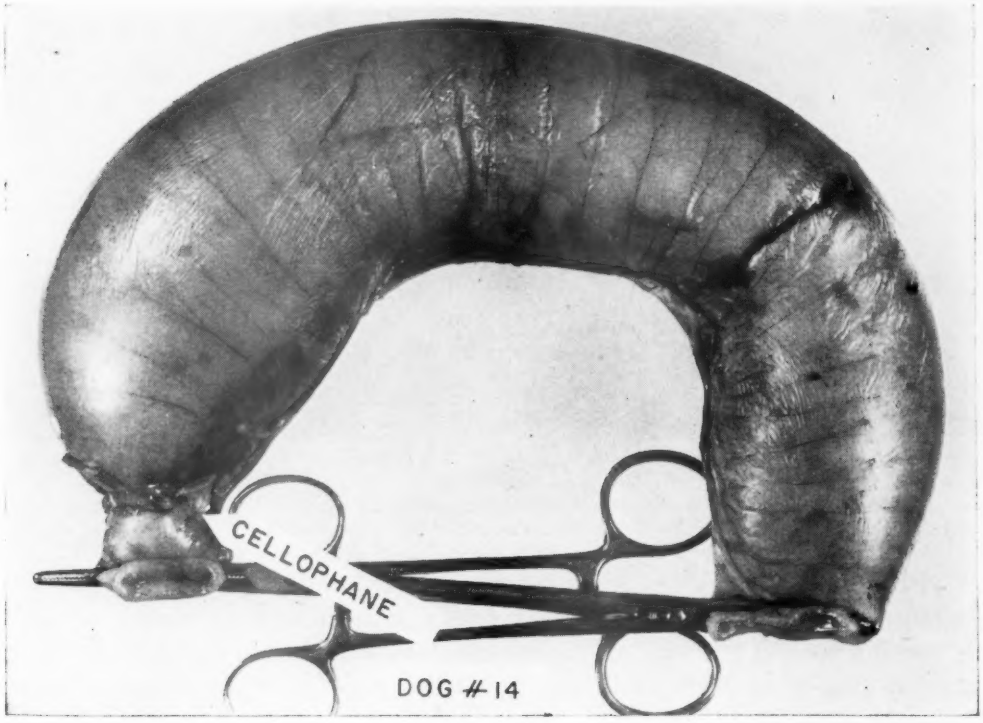

Fig. 2.—Resected bowel showing dilatation and hypertrophy proximal to obstruction with distal collapse.

In 13 of the 16 animals studied completely, a high degree of obstruction was produced, with marked proximal dilatation and hypertrophy, and lumina less than 6 mm. in diameter. The other 3 animals had definite obstruction which was classified as minimal, with lumina at the obstructive site ranging between 1 and 3 cm. In 2 of the latter animals there was a small amount of barium still noted in the terminal ileum on the twenty-four hour film, while in the third, the barium was still in the colon at twenty-four hours. Several nonobstructed dogs were given barium for comparison, and in each of these, no barium remained in the small bowel at twenty-four hours, and in only 1 animal was barium demonstrated in the colon at that time. The majority of the x-ray films taken of the other 13 dogs twelve and twenty-four hours after administration showed stasis of barium in the dilated terminal ileum, but with two exceptions, as the animals were followed over a period of time, the barium was gradually evacuated. Two dogs were followed for five days, during which time almost all of the barium remained in the terminal ileum, but at autopsy the intestinal contents containing barium were in a liquid state and there was no evidence that obstruction had been completed. Increasing distention or vomiting was not noted after barium administration. In 11 animals, the contents of the proximal dilated bowel at autopsy were in a fluid state, but were classified as semisolid in 5. It is particularly significant that complete obstruction did not develop in any instance.

A. *B.* *C.*

Fig. 3.—Series of films showing progressive dilution of barium. *A*, Twenty-four hours; *B*, forty-eight hours; *C*, eighty-four hours.

In those animals in which there was marked stasis of barium in the bowel proximal to the obstruction (Fig. 3), as the animals were followed, even if little barium passed the site of obstruction, the barium became less dense, presumably due to an increase in fluidity of the intestinal contents and "scattering" of the barium.

DISCUSSION

These experiments demonstrate clearly that barium does not form a puttylike impacted mass when stasis occurs proximal to obstruction in the small bowel, nor does it render a partial obstruction complete. It is possible that the fluidity of the intestinal contents is later increased due to the mechanical stimulation of the presence of the barium. Unquestionably, the stasis proximal to obstruction is temporarily increased by the presence of the barium, but this should have little deleterious effect. The administration of barium by mouth to patients with chronic partial small bowel obstruction (after obstruction of the colon has been ruled out) should be safe and, by the manipulations of the radiologist and his fluoroscopic observations, might result in a fairly high percentage of correct radiologic diagnoses. However, the administration of barium in cases of complete obstruction, or in cases of acute obstruction due to adhesions, as described by Cantor and others,[2] or due to incarcerated hernia or volvulus, seems unnecessary, since the diagnosis should be made on the basis of clinical findings and scout films. It is also unwise, because unless the barium is evacuated before operation, it will interfere with the resumption of intestinal motility during the postoperative period.

SUMMARY

A satisfactory method for producing partial high-grade intestinal obstruction in the experimental animal has been presented. Animals prepared in such a way were studied by x-ray films at varying intervals after the administration of barium through a Levin tube. The animals then were autopsied, and the character of the bowel contents and size of the lumen at the site of obstruction were correlated with the x-ray findings. It was noted that complete obstruction due to impaction of barium did not occur and, in most cases, the barium remaining in the small bowel was in a fluid state.

CONCLUSIONS

Under the conditions of these experiments, barium failed to produce complete obstruction after entering the small bowel proximal to a previously produced partial obstruction.

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THE SIGNIFICANCE OF LOCAL VASCULAR CHANGES IN BLEEDING PEPTIC ULCER

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DESPITE the many advances and additions to the armamentarium in the conservative treatment of peptic ulcer, including various antacids and dietary regimens, as well as the newer anticholinergic drugs and the psychosomatic approach to the problem, chronic peptic ulcer remains a major disease, recurrences are frequent, and complications, including hemorrhage, are not uncommon.

It has been noted by Wangensteen[1] that evidence of peptic ulcer can be found in one of every twenty autopsies that are carefully performed; moreover, 10 per cent of all ulcer patients enter the hospital because of hemorrhage. Herbut[2] estimated that 19 per cent of all peptic ulcer patients have one or more hemorrhages, and Eusterman[3] has indicated that of all cases of gross hemorrhage from the upper gastrointestinal tract, peptic ulcer is responsible at least 85 per cent of the time.

While the reasons usually alluded to in explaining the failure to produce a permanent cure with medical therapy are inadequate dietary or pharmaceutic routines, poor cooperation ("patient failure"), or ulcer diathesis of the patient, the importance of the pathologic changes in the ulcer and surrounding area must not be overlooked. It is readily understandable why, after replacement of the muscle layers and submucosa in chronic ulcer, prevention of coaptation of the ulcer edges occurs, healing is slow, and a favorable nidus for recurrence is produced. In addition, as emphasized by Boyd,[4] inflammation, as evidenced by dilated vessels and foci of inflammatory cells, continues to occur in the ulcer bed long after the scar tissue appears quiescent and regeneration of the superficial mucosa has taken place.

Vascular changes in the local ulcer area are well described in many textbooks of pathology and treatises on the subject, but the significance of these changes, particularly in event of erosion of the vessels by the ulcer, has received little consideration. The decision for surgical intervention in the case of hemorrhage from peptic ulcer is usually based (far too often) on the degree of generalized arteriosclerosis which the patient has, the common denominator being the patient's age. It is the purpose of this study to review the local vascular changes in cases of peptic ulcer in which patients have had severe hemorrhage, in an attempt to determine whether these changes make severe hemorrhage more likely in case the vessel(s) becomes eroded by the ulcer, regardless of the presence of, or lack of, generalized sclerotic vessels.

Received for publication, Oct. 2, 1953.

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GENERAL CONSIDERATIONS

Besides the hemodynamics and clotting power of the blood, there are obviously local factors that determine whether the blood loss in bleeding peptic ulcer will be severe. Among these may be mentioned the size or number of vessels eroded, the integrity of the neuromuscular system, and pathologic changes in the vessels which might affect contractility or smooth coaptation of the vessel intima. Anatomically, although the vessels commonly involved in fatal or severe hemorrhage from peptic ulcer are the right or left gastric arteries, or the gastroduodenal or their branches[5] (especially the superior pancreaticoduodenal), severe and even fatal bleeding may occur from the erosion of small vessels entering the ulcer bed, which may not be grossly discernible at surgery or at autopsy. Occasionally, the exact bleeding point may not be seen and may not be defined unless careful serial sections for microscopic examination are made. It is also of some importance to recall that the pylorus, although frequently the site of peptic ulcer, is rarely, if ever, the site of severe bleeding because of its relative avascularity.[6]

According to Anderson,[7] localized infections in tissues may involve an artery to produce an acute periateritis. Such a vessel may become thrombosed or weakened to the point of rupture. As a matter of fact, Lisa and Likely[8] reported cases of chronic peptic ulcer with death from exsanguinating hemorrhage in which bacterial infection of the wall of the involved vessel was demonstrated in each case. Bacterial involvement of the vessels in bleeding ulcer, however, is rarely elicited in the pathogenesis of severe hemorrhage, and was not seen in any of the cases in the present study.

THE AGE FACTOR

In most large series of cases reported, fatalities from bleeding peptic ulcer occur most frequently in the age group 45 to 60 years.[9-11] Bockus[12] reported no deaths in the 20- to 30-year age group, while Methany and Green[13] went so far as to say that bleeding ulcer in young people is a self-limited and nonfatal disease, with favorable results to be expected with conservative therapy in all cases of patients under 45 years. Such optimism is not universally held, and to make such optimism the basis for treatment is to invite disaster.

Meyer and his associates[14] reported a mortality rate of 30 per cent in patients under 45 years and 43 per cent over 45 years of age treated surgically, as compared to no mortality in the age group under 45 years and 21 per cent over 45 years treated medically. However, in evaluating any statistics regarding therapy, certain considerations must be entertained before any conclusions are made; among these we must include other factors in addition to concomitant disease processes, particularly in regard to the ability to withstand blood loss. Also, an important fact to keep in mind always in attempting to compare treatment is the amount of blood lost before surgery is finally undertaken. In hemorrhage from any source, the surgical risk rises with each hour surgery is withheld.

That age per se should not be the sine qua non in determining whether surgery should be resorted to in severely bleeding peptic ulcer is further attested by the fact that severe and fatal hemorrhage may occur in infants and children. Bird and his group[15] have reported twenty-two cases of bleeding peptic ulcer in the newborn infant. Rothwell and Strode[16] reported a case of an infant with continued bleeding from gastric ulcer in which a lifesaving gastric resection was performed.

METHODS AND FINDINGS

Twenty-five cases of bleeding peptic ulcer were reviewed, particular attention being paid to the vascular changes in the ulcer bed and immediate surrounding area and, in addition, to the morphology of the gastric and duodenal vessels remote from the ulcer area. In the fatal cases, a search for arteriosclerosis or vascular disease in other organs and tissues was made.

TABLE I. CASE SUMMARIES

CASE NO.	AGE (YR.)	ULCER LOCATION	HEMATOCRIT	TREATMENT	RESULTS	VASCULAR PATHOLOGY IN SURGICAL OR AUTOPSY SPECIMENS
1-A1621	29	Duodenum	18	Medical	Died	Marked intimal proliferation of vessels in ulcer bed; no arteriosclerosis in coronaries or aorta (Figs. 1 and 2)
2-S2887-52	63	Stomach	20	Surgical	Lived	Irregular thickening of intima of vessels in ulcer base; elsewhere evidence of arteriosclerosis in vessels remote from ulcer area (Fig. 3)
3-S1964-50	49	Stomach	24	Surgical	Lived	Obliterative endarteritis in some vessels in ulcer base; elsewhere vessels show intimal proliferation (Fig. 4)
4-S11992	29	Stomach	21	Surgical	Lived	Replacement of smooth muscle by hyaline fibrous tissue; thickening of intima (Fig. 5)
5-A2000	45	Duodenum	21	Medical	Died	Large vessel (superior pancreaticoduodenal) was eroded by ulcer; artery had "pipe-stem" appearance; intimal proliferation (Fig. 6)
6-S1271-50	47	Duodenum	20	Surgical	Lived	All vessels in ulcer bed were thickened; no large vessels in ulcer area
7-S2231-52	49	Stomach	22	Surgical	Lived	Marked intimal proliferation of all vessels in ulcer area; obliteration of elastica interna
8-S3203-52	54	Stomach	24	Surgical	Lived	Irregular intimal thickening; invasion of vessel walls by inflammatory cells
9-S40-53	62	Duodenum	20	Surgical	Lived	Vessels in ulcer bed and adjacent area show intimal proliferation; in some cases there is active fibrosis (proliferation of fibroblasts) in the media; distally from the ulcer site the vessels appear normal
10-S23751-52	26	Duodenum	16	Surgical	Lived	Small vessels in ulcer bed have thickened walls; vessels elsewhere appear normal

The cases (ten of which are summarized in Table I) fulfilled the following criteria: (1) Severe bleeding had occurred, based on a hematocrit of 25 per cent or less. (2) There was no history of hypertension or renal disease. (3) No noteworthy generalized arteriosclerosis was demonstrable on examination, at surgery, or on examination of vessels at autopsy. (4) No history of cardiac disease was elicited and none was evident at necropsy. (5) Blood vessels in the ulcer area were demonstrable in the microscopic sections.

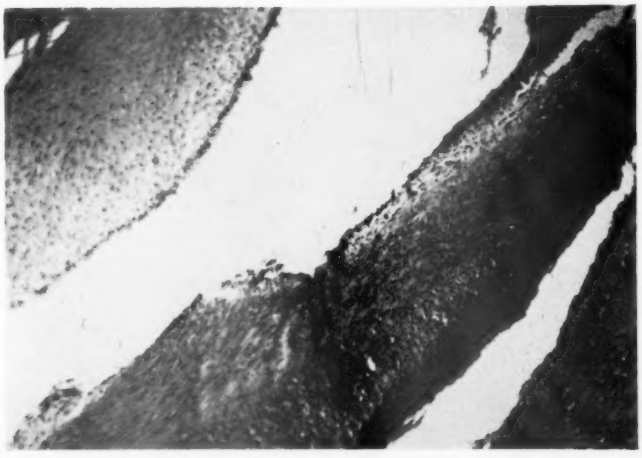

Fig. 1.—Case 1. Section of artery at site of erosion showing infiltration of inflammatory cells.

Fig. 2.—Case 1. Same vessel as in Fig. 1 remote from site of erosion. Note marked intimal proliferation.

It was found from these studies that the pathologic changes within the blood vessels were similar regardless of the age of the patient. These changes were: (1) proliferation of the intima, (2) replacement of smooth muscle of the media by fibrous tissue, and (3) varying destruction of the elastica interna. For details see Table I.

All the patients here reported had severe hemorrhage. In one-half the patients, it was the first episode of bleeding, which correlates with the findings

of Shapiro and Shiff,[17] who reported that no significance in the severity of bleeding could be attached to the fact that hemorrhage had or had not occurred one or more times previously, or whether the present episode was the initial one.

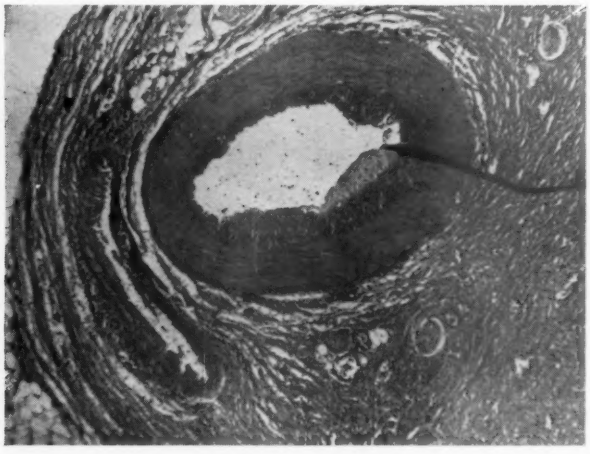

Fig. 3.—Case 2. Vessels just beneath ulcer. Both artery and vein show irregular thickening of the intima.

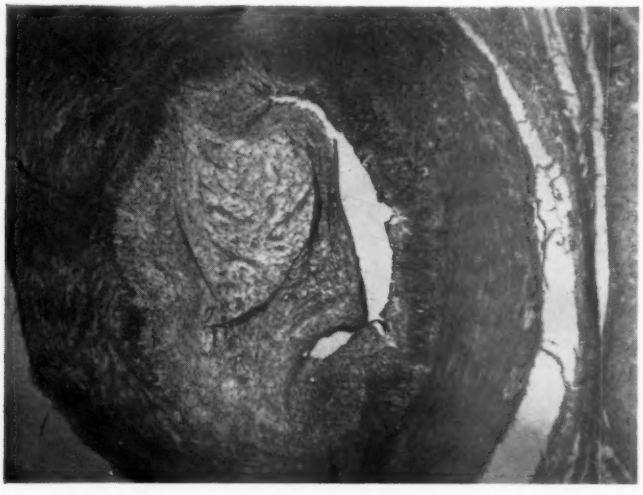

Fig. 4.—Case 3. Artery in ulcer bed showing obliterative endarteritis.

Significant also in the treatment of bleeding peptic ulcer is the possibility of recurrence of hemorrhage. Holman,[18] for example, found that of those patients who recover under conservative management, more than 50 per cent bleed again; moreover, at least one-half the patients who continue to bleed forty-eight hours after being placed on a strict medical regimen expire.

It will be noted from among the ten cases selected in this study for summary that in three of the cases, patients were under 30 years of age. The pa-

tient in Case 1 died from exsanguination after several days of intensive medical management, whereas the patients in Cases 4 and 10 were treated surgically and survived. That age, per se, may not be the important factor in ability to survive severe bleeding is also indicated by the studies of Bohrer,[19] who found a mortality rate of 20 per cent in patients under 45 years.

Fig. 5.—Case 5. Gross specimen showing artery from which fatal hemorrhage occurred. Microscopically, the vessel was thickened.

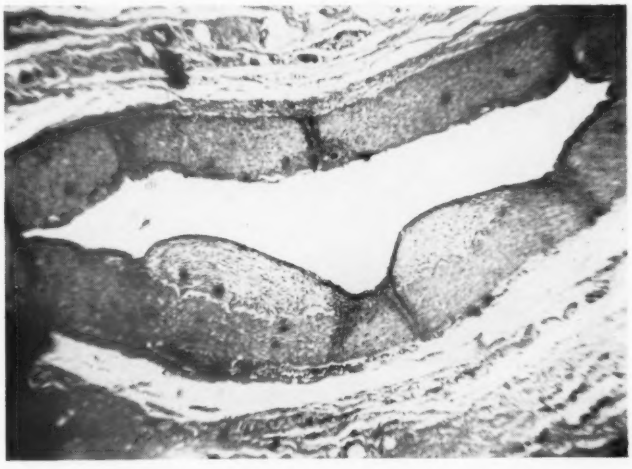

Fig. 6.—Case 7. Note beginning of intimal proliferation. Other vessels in the ulcer bed showed marked proliferation of the intima.

The surgical procedure in the cases selected in this study consisted of subtotal gastric resection of the Polya type, after immediate stoppage of the bleeding point by ligation of the major vessel, if possible, or plication of the ulcer over the vessel(s). Massive replacement of blood was attempted in each case, an average of 2,500 c.c. being given before operation was performed.

In one patient (Case 5) treated medically, 7,500 c.c. of blood was given before he expired. At autopsy, as in the other cases here selected, there was no generalized arteriosclerosis, the significant vascular changes being limited to the ulcer area.

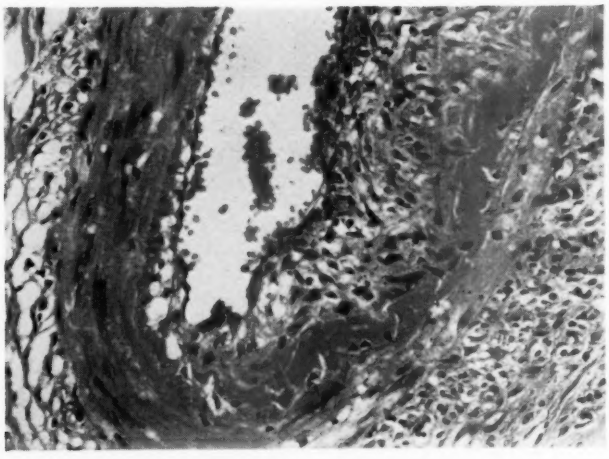

Fig. 7.—Case 4. The muscular media has been partially replaced by fibrous tissue.

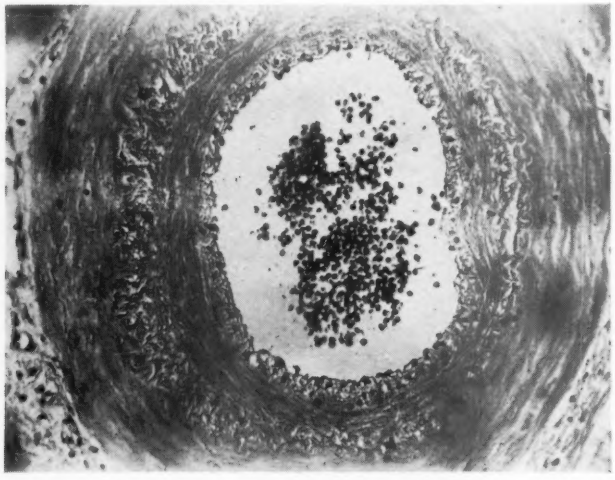

Fig. 8.—Case 8.—There is intimal proliferation, fibrosis of the media, and preservation of the elastica interna.

In a comprehensive review of severely bleeding peptic ulcer, Lewison[20] has emphasized the fact that hematemesis is indicative of severe hemorrhage, while melena alone may not be. Although some idea of the amount of blood loss may be obtained by estimating the quantity from hematemesis or melena, it must be remembered that a large volume of blood may be within the gastrointestinal tract concealed from the observer. In addition, of course, the volume of packed red blood cell determination, blood volume, red cell count, and hemoglobin all vary depending on the shift of fluid from the intracellular

to extracellular space as bleeding progresses, and these laboratory determinations at the moment may not be entirely accurate in giving the exact picture of continued or severe blood loss. Repeated, frequent determinations, as well as careful clinical observation and cooperation between internist and surgeon, are essential in proper management.

The true indication of the severity of bleeding must be individualized, and it is that amount of blood necessary to restore the physiologic hemodynamics. Because of this fact, it has been suggested by Clark[21] that each case of bleeding peptic ulcer be managed as though preparing for surgery, conservative management being continued only if hemodynamics is restored by a given volume of whole blood (such as 4 or 5 units) and is maintained thereafter without further transfusions.

CONCLUSIONS

The reason for the increased mortality rate in the older individual with severely bleeding peptic ulcer, other factors being equal, has usually been attributed to arteriosclerosis of the vessel(s) eroded by the ulcer and to definitive surgical treatment being arbitrarily withheld in the patient with supposed normal, elastic blood vessels—age, a priori, being made the common denominator in conservative versus surgical management. Oöphuls[22] believed that arteriosclerosis in peptic ulcer is frequently observed and may occur as a local process in young persons. Askanazy,[23] on the other hand, observed that gross arteriosclerosis of the vessels of the celiac axis is uncommon in patients even in the fifth or sixth decade.

In the cases reviewed in this study, there was no arteriosclerosis of the vessels to the stomach or duodenum peripheral to the ulcer area. It is suggested that the vascular changes seen in peptic ulcer are secondary to inflammatory processes in the ulcer bed, and the changes seen depend on the stage seen, which, in turn, depends on the extent and duration of the inflammatory reaction, regardless of the age of the patient or vascular pathology elsewhere.

The pathologic microscopic changes included invasion of vessels by inflammatory cells, irregular proliferation of the intima (which at times was striking), replacement of smooth muscle of the media by fibrous tissue, and inconstant dehiscence of elastic fibers. It may be conjectured that the pathologic physiology may be similar to arteriosclerosis and, in the case of erosion, bleeding from the vessel is likely to be severe due to impaired contractility and poor coaptation of the intima, the former being due to replacement of smooth muscle by fibrous tissue, and the latter due to irregular intimal proliferation. Furthermore, if obliterative endarteritis occurs, it may be supposed that the resulting ischemia to the area may further delay healing of the ulcer, further scarring, and make recurrences more likely—all of which impair the success of conservative therapy.

SUMMARY

Twenty-five cases of severe hemorrhage from peptic ulcer were reviewed and ten cases were selected for summary which fulfilled the following criteria:

(1) The hematocrit was 25 per cent or lower during bleeding. (2) No severe concomitant disease was present. (3) Microscopic sections of the surgical specimen or autopsy tissues were made showing blood vessels in the ulcer area (serial sections).

No arteriosclerosis of the gastric or duodenal vessels was demonstrated. The vascular changes occurring in the vessels in the ulcer area were similar, regardless of the age of the patient, and consisted, in the main, of proliferation of the intima (irregular), varying replacement of the muscular media by fibrous tissue, inconstant changes in the elastica interna, and, in some cases, active inflammation of the vessels with invasion of the vessel walls with inflammatory cells.

Vascular changes in peptic ulcer are well known and have been frequently described; however, the significance of these changes has received little consideration, the decision for surgical treatment in the event of hemorrhage too often being based on the supposed degree of arteriosclerosis present, the common denominator being the age of the patient.

It is suggested that the pathologic changes in the vessels in peptic ulcer are secondary to the inflammatory process in the ulcer area, and that these changes render the vessels less elastic, impair their contractility, and prevent smooth coaptation of their intima; thus, in case of erosion, bleeding is likely to be severe, depending on the size or number of vessels involved. Inasmuch as these vascular changes occur regardless of the age of the patient, it is proposed that making merely the age of the patient the sine qua non in determining therapy is not valid.

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ONE-STAGE PANCREATODUODENECTOMY WITH RESECTION OF THE SUPERIOR MESENTERIC VEIN

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ALLEN Whipple, in 1935, reported on three patients with carcinoma of the ampulla treated by a two-stage radical pancreatoduodenectomy, and stimulated further surgical attack on the problem of biliary tract carcinomas.

Subsequently, many reports of technically successful resections of ampullary and pancreatic neoplasms were added to the surgical literature. Reports of encouraging numbers of patients with ampullary carcinoma who, subsequent to operation, survived five years or more have been published.[2, 3] These results should be achieved as predicated by the autopsy studies of Baggenstoss, who found that in over one-third of the cases no metastases were present.

Unfortunately, a similar encouraging situation does not exist in regard to carcinoma of the pancreas. Nevertheless, increasing numbers of five-year survivals are being reported. The modified pancreatoduodenectomy procedures described herein are further attempts to achieve a satisfactory "cancer operation" in those patients in whom the disease is still localized. Descriptions of similar operations have been published.[5, 7, 9] Certainly such operations will not result in a substantial impress upon this dread disease, but in an occasional case they may mean the difference between death and survival.

A brief review (Table I) of patients with ampullary and pancreatic carcinomas between 1936 and 1951 seen at the University of Minnesota Hospitals is included to emphasize the scope of the problem at a medical center.

All cases with a definitive diagnosis of cancer of the biliary tree and pancreas are included; however, it must be remembered that a certain additional number of patients were briefly seen in the outpatient department and discharged as hopeless cases without establishment of a diagnosis. Second, undoubtedly others with this disease were considered hopeless cases by their physicians and were not referred to the University Hospitals for treatment. Therefore, the data included in graphic form cannot be interpreted as accurately reflecting the outcome of a wholly unselected group of patients with this disease.

Note that a curative procedure was attempted in only 15.4 per cent of the cases, including many extremely aggressive procedures.

Received for publication, Sept. 29, 1953.

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TABLE I. CANCER OF BILIARY TREE AND PANCREAS (1936-1951)

Total cases	486	
Surgery	333	
Exploratory only		85
Palliation		173
Resection		75

CARCINOMA OF PANCREAS

There were 193 patients with carcinoma of pancreas. In 145 patients, the lesion occupied the head of the pancreas, and in 48, the body or tail.

The age incidence of these patients is depicted in Fig. 1.

It is interesting to note the predominance of men afflicted with pancreatic cancer (and carcinoma of the common duct) in contrast to the preponderance of women afflicted with carcinoma of the gall bladder (Fig. 2).

The most frequent symptoms mentioned by patients with carcinoma of the pancreas at the onset of the disease were pain 86 per cent, jaundice 29 per cent, and anorexia 15 per cent. Other prominent symptoms were diarrhea and constipation, nausea, vomiting, bloating, and belching. The duration of symptoms is indicated in Fig. 3.

Both patients with lesions of the head of the pancreas and those with lesions in the body and tail indicated that their pain was in the mid-epigastrium. Back pain, which has been considered an important diagnostic symptom, was elicited in one-third of those with lesions of the body and tail and only 6 per cent of those with tumors in the head of the pancreas. The complaints at the time of admission are summarized in Fig. 4.

Excessive weight loss has been noted by most clinicians and was confirmed in this series of patients (Fig. 5).

Despite an aggressive surgical attitude toward pancreatic cancer in the last few years, only 20 of the 193 patients were subjected to radical resection because of local extension or widespread metastases (Table II).

TABLE II. CARCINOMA OF PANCREAS (1936-1951)

Total cases	193	
No surgery	63	
Surgery	130	
Exploration		35
Palliation		75
Resection		20

Survival of these patients is extremely discouraging, as is indicated graphically in Table III.

TABLE III. SURVIVAL OF PATIENTS WITH CARCINOMA OF PANCREAS

	TOTAL	ONE MONTH	TWO MONTHS	SIX MONTHS	ONE YEAR
No surgery	63	22 (35%)	10 (16%)	5 (8%)	0
Exploration	35	12 (34%)	6 (17%)	1 (3%)	0
Palliation	75	47 (63%)	34 (45%)	7 (9%)	3*
Radical	20	14 (70%)	11 (55%)	4 (20%)	0

*Insertion of T tube, 2 cases; choledochojejunostomy, 1 case.

It must be remembered that with such a small number of cases, individual percentages are not valid and are included only to indicate the trend of survival with and without surgery.

PALLIATION

It is difficult to provide effective palliation for this tumor. Certainly, the patient and relatives are relieved to see the disappearance of jaundice and its attendant discomfort. This can be most simply provided by insertion of a T tube, but this technique has the disadvantages of loss of electrolytes and water, and the problem of continual external drainage. We prefer, if choledochojejunostomy is impossible, to insert an enterostomy tube also, which can be connected to the T tube externally, allowing the bile to be returned to the intestine. The enterostomy tube can, of course, also be used for feeding purposes.

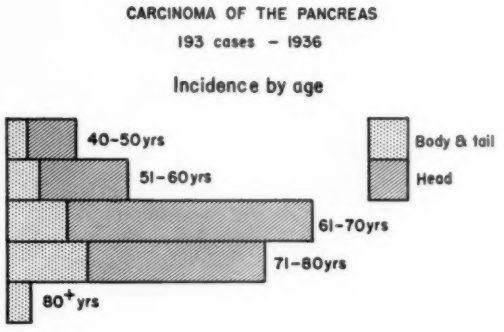


Fig. 1.

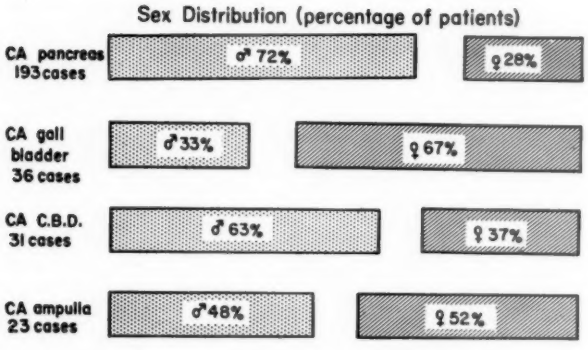
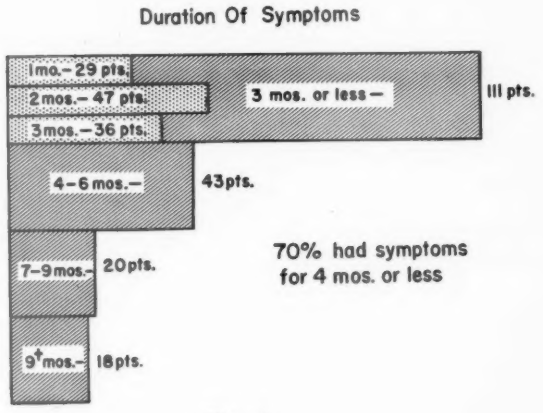


Fig. 2.

Although choledochojejunostomy is increasingly popular, our experience does not indicate that it offers an increased period of palliation. Both the loop of jejunum and the Roux Y principle have been utilized, with little difference clinically.

Finally, it should be noted that radical pancreaticoduodenectomy is not a satisfactory palliative procedure and should be used only when there is a possibility of effecting a curative excision.



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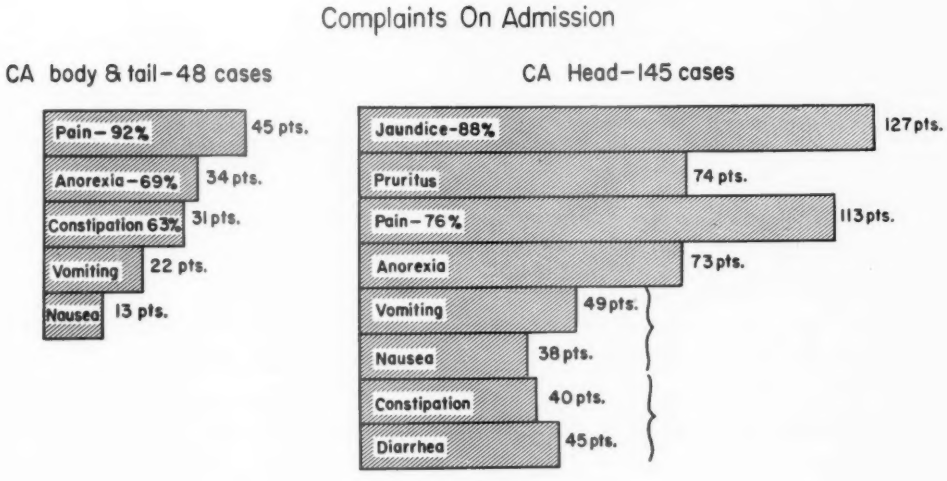

Fig. 4.

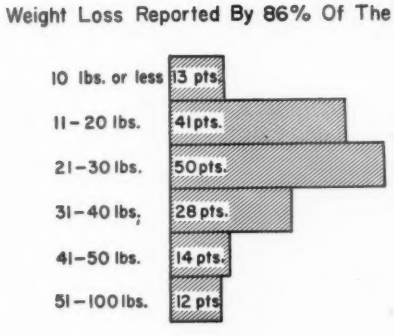

Fig. 5.

LONG-TERM SURVIVORS

Five-year survivals following resection of biliary and pancreatic neoplasms in this hospital number only two patients with carcinoma of the ampulla and one with a diagnosis of sarcoma of the body of the pancreas. All three patients are in good health.

In this regard it should be noted that a patient with pancreatic carcinoma (not included in this study) operated upon by Dr. Clarence Dennis, formerly on the University of Minnesota staff, is still alive and well. This woman was 70 years of age when first admitted to the Minneapolis General Hospital in 1943 with severe cardiac decompensation, auricular fibrillation, generalized arteriosclerosis, and hypertension. On March 28, 1945, a pancreaticoduodenostomy was performed and the patient was discharged on May 4, 1945.

The carcinoma was described as being about 3 cm. in diameter; no involved lymph nodes were noted. This patient has since survived many bouts of cardiac decompensation, and at last report was alive (eight years) without recurrence of the cancer.

EXTENSION OF THE PANCREATODUODENECTOMY PROCEDURE

In recent years, several surgical groups have reported their attempts to extend the standard one-stage Whipple procedure. Child has reported several two-stage pancreatoduodenectomies with portal vein resection,[5] and Parsons[9] and McDermott[6] have reported one-stage operations. In several of the latter cases, however, it was noted that the portal vein was partially occluded prior to resection.

Child[4, 5] carried out a series of excellent experimental and clinical studies to demonstrate the feasibility of interrupting the portal vein. Finally, he reported a successful two-stage pancreatoduodenectomy with resection of an unobstructed portal and mesenteric vein. Such a procedure makes possible an attempt to perform a curative procedure in those patients with a carcinoma of the head and/or uncinate process with extension to the portal vein. Interestingly enough, examination of our specimens with extension of the tumor to the vein did not show actual penetration of the tumor through the vein wall.

A patient with carcinoma of the pancreas and involvement of the superior mesenteric vein but without significant reduction of the lumen necessitating resection of the vein has been previously reported by us.[7] Briefly, the tumor was found to be adherent to the superior mesenteric vein and, therefore, after gently clamping both the superior mesenteric vein and artery, the pancreatic dissection was completed, a 3 cm. section of the vein resected, and reanastomosis performed. The vessels were clamped for ninety-five minutes without ill effect. Although the patient enjoyed an uneventful postoperative course, she was readmitted forty-two days later with evidence of widespread metastasis and died afterward. Autopsy examination revealed the vein anastomosis to be widely patent.

A second such operation with excision and reanastomosis of the superior mesenteric vein was performed by Dr. D. Ferguson and one of us without event. The superior mesenteric vessels were clamped for forty-three minutes (Fig. 6, *3*) without interruption of the artery.

In a third patient in whom the carcinoma was adjacent to the mesenteric vessels, reanastomosis was impossible because of the length of the involved segment. The superior mesenteric vein was dissected free of several small branches and then anastomosed end to side to the vena cava (Fig. 6, *2*). The mesenteric vessels were simultaneously clamped for forty minutes.

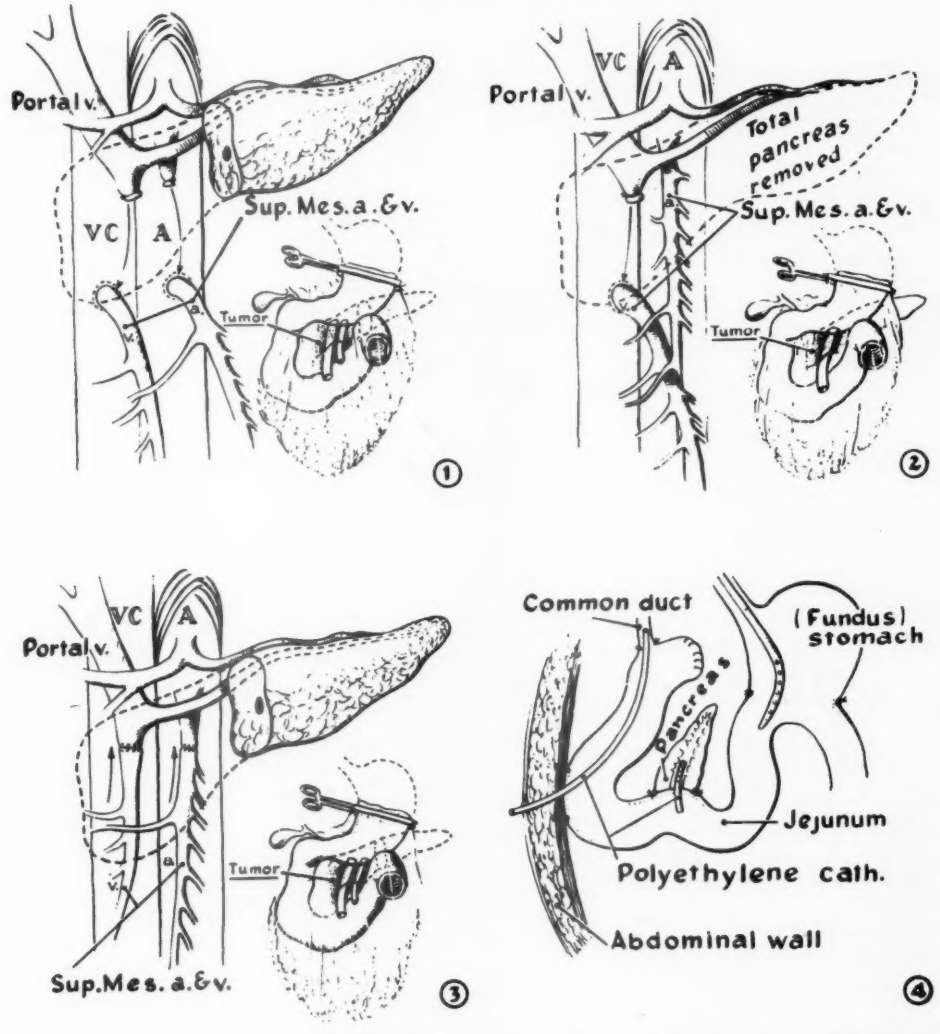

Fig. 6.—Possible modifications of radical pancreatoduodenectomy when the superior mesenteric vessels are involved by tumor.

Clinically, the tumor seemed to extend along the pancreatic duct into the body of the pancreas and, therefore, a total pancreatectomy was performed. The postoperative course was complicated by an inability to control adequately the blood sugar levels and, consequently, a normal electrolyte pattern. Several

episodes of profound hypoglycemia were followed by a continuing comatose condition. The patient died thirty-six days subsequent to the operative procedure.

Post-mortem examination revealed the anastomosis of the superior mesenteric vein to the vena cava to be patent. No evidence of residual tumor was found.

Fig. 6, *4* illustrates an operative technique somewhat different from those described in the literature which is useful in special instances. The common duct should always be divided as high as possible, usually above the level of the cystic duct, since experience has shown that intramural and extramural lymphatics of the common duct are commonly involved. Division of the common duct at or immediately below the apparent junction should be avoided. Often the cystic duct forms an integral part of the common duct with a slightly reduced diameter of the common duct or a double-barreled configuration which can make the choledochojejunostomy more difficult. If the surgeon wishes to splint the anastomosis temporarily, a minute polythene tube can be used, as illustrated. Usually this technique is not necessary. Similarly, a small polythene tube with multiple perforations, as recommended by Dennis and Varco, may be used to splint the pancreatic duct jejunal anastomosis temporarily. A mucosa-to-mucosa anastomosis with a single row of interrupted sutures of 00000 silk is preferable. As has been pointed out many times, an adequate gastric resection should be peformed in order to obviate the subsequent development of marginal ulcers. This complication has been seen even in patients surviving only a short time.

Obviously, the surgical approaches outlined have a limited usefulness. The percentage of pancreatic cancer victims dying with the tumor still limited to the local area is unknown and awaits a study similar to that concerning ampulla carcinoma reported by Baggenstoss.

Further, it is evident that the rapacious character of this tumor is such that even wide dissections of the definitive tumor may fail to eradicate the tumor completely, as is amply indicated by the dearth of five-year survivals following radical surgery. This is not intended to imply that further attempts to treat this tumor surgically should be abandoned, but is merely a recognition of the severity of the problem.

DISCUSSION

The problems attendant on resection of the superior mesenteric vein are:

(1) increased operative hemorrhage from ligation of the portal vein (Child),

(2) thrombosis of the portal system resultant to temporary occlusion of the mesenteric vessels,

(3) hypoxia with subsequent injury to the intestine following prolonged clamping of the mesenteric vessels, and

(4) re-establishment of the portal circulation or performance of a portal canal shunt.

As is indicated in our abbreviated case reports, none of these problems preclude the successful accomplishment of a one-stage procedure.

In contrast to the experience of Child, no increased portal hemorrhage was encountered with temporary clamping of the portal vein during dissection even though in none of our cases was the portal vein obstructed or its lumen significantly reduced. It is interesting that McDermott, in his summary of previous clinical and experimental observations, stated that "resection of the portal vein together with the pancreas and duodenum is possible, but only after collateral channels have been developed."[6] His own excellent case report of a one-stage radical pancreaticoduodenectomy with segmental resection of the portal vein and its anastomosis to the vena cava did not indicate an unusual degree of hemorrhage; narrowing of the portal vein was not mentioned.

In regard to engorgement of the portal system during surgical treatment of the superior mesenteric vein, it is imperative that the accompanying artery be occluded simultaneously or preferably a few seconds earlier.

The threat of mesenteric thrombosis following prolonged clamping of the superior mesenteric vessel for intervals necessary to complete a block dissection of the pancreas and reanastomosis of the vessels cannot be disregarded. Fortunately, such a catastrophe was not encountered in our patients nor has it been reported by others. Undoubtedly, the fact that most patients with carcinoma of the pancreas have an obstructed biliary tract and some depression of liver function increases the margin of safety.

Experimental studies by Nelson and Kremen[8] indicated that, in the dog, either the superior mesenteric artery or vein could be safely clamped for two hours whereas occlusion for four hours was fatal in a majority of the animals. Occlusion of the superior mesenteric vein alone definitely was more lethal. They reported that preoperative administration of heparin significantly extended the period during which the vessels could be occluded.

Finally, the possible patterns of vessel resection are several. Fig. 6 depicts the procedures performed at this clinic and, in addition, a proposed scheme of resecting both the superior mesenteric vessels with their respective anastomosis to the portal vein and aorta. Similarly, it might be necessary and advisable to resect the portal vein proximal to the entrance of the splenic vein with excision of the splenic vessels and the accompanying lymphatics.

SUMMARY

1. Partial statistics concerned with diagnosis and treatment of biliary tract malignancy are presented, and several long-term survivals noted.

2. The technical feasibility of resecting the superior mesenteric vessels in order to accomplish a more adequate resection of the pancreas is demonstrated.

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FEMORAL HERNIOPLASTY: SUPRAPUBIC EXTRAPERITONEAL (CHEATLE-HENRY) APPROACH

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THE surgical correction of a femoral hernia has generally been performed through a groin incision placed either above or below the inguinal ligament. A combination of the two incisions is occasionally employed. There is no doubt but that an adequate hernioplasty can be performed through either approach, although a strangulated femoral hernia is usually better managed through an incision above the inguinal ligament. The purpose of this presentation is to emphasize the advantages of a seldom-used alternative approach and to suggest an improved technique for the actual hernioplasty.

The approach to be described is initiated by a suprapubic, abdominal incision, either vertical midline or Pfannenstiel. The incision is carried through the fascia, and from this point the dissection remains extraperitoneal and the femoral region is approached from above, behind the rectus muscle.

The advantages of the suprapubic extraperitoneal approach for the repair of femoral hernia have been stressed in several reports.[1-5] It has recently been referred to as the "Henry Operation" in the belief that Henry[3] in 1936 was the first to describe it. However, credit should be given to Cheatle for the basic concepts of this approach. In 1920,[1] he reported such a repair of a femoral hernia through a rectus muscle-splitting extraperitoneal approach on the involved side. He later[2] reported the use of a midline abdominal incision for the repair of bilateral hernias, inguinal as well as femoral.

When the suprapubic extraperitoneal approach has been used, closure of the femoral ring has been accomplished by a variety of methods. These methods have included plugging of the femoral canal with a segment of saphenous vein, elevation to Poupart's ligament of a flap of periosteum from the superior pubic ramus, or of a flap of fascia overlying the pectineus muscle, and, more commonly, simple suture of Poupart's ligament to Cooper's ligament. The hernioplasty technique presented here utilizes a doubled layer of the fused transversalis fascia and aponeurosis of the transversus muscle which is anchored to Cooper's ligament.

TECHNIQUE

The accompanying illustrations were obtained from a cadaver preparation. An actual femoral hernia is not present in this specimen, but this is not necessary for adequate illustration of the repair. The dissection demonstrates the superior exposure and the resultant ease with which the hernioplasty is

Received for publication, Oct. 5, 1953.

accomplished. The photographs are presented in serial arrangement and with their labels are self-explanatory.

DISCUSSION

During the four-year period from July, 1949, to July, 1953, 214 patients with femoral hernias were operated upon at the Los Angeles County Hospital.

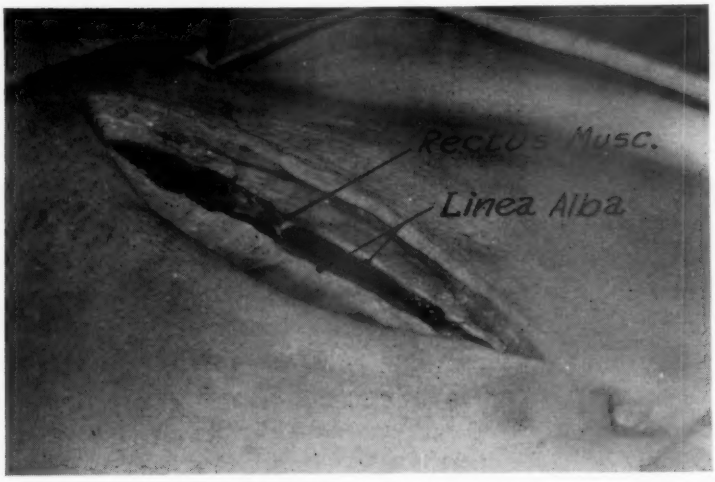

Fig. 1.—Either a vertical midline or Pfannenstiel incision may be used. Each affords equally good exposure.

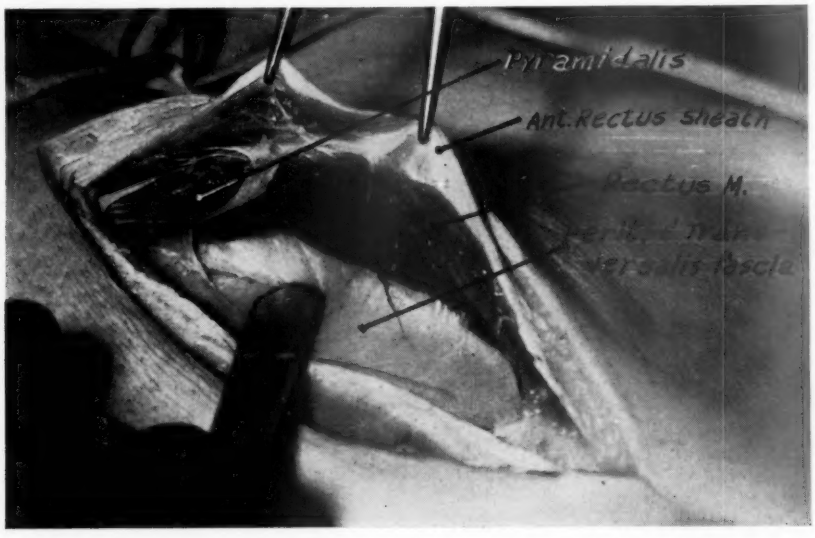

Fig. 2.—Beginning extraperitoneal dissection. This is accomplished bluntly by finger or gauze dissection.

Of this number, 113 were operated upon through the suprapubic extraperitoneal approach. No patient has returned for treatment of recurrence. This method is now preferred by the majority of the resident and attending staff of this hospital.

This approach has the obvious advantage of allowing the performance of a bilateral hernioplasty through one incision, a factor of importance since about 20 per cent of femoral hernias are overtly bilateral. We feel, however, that its

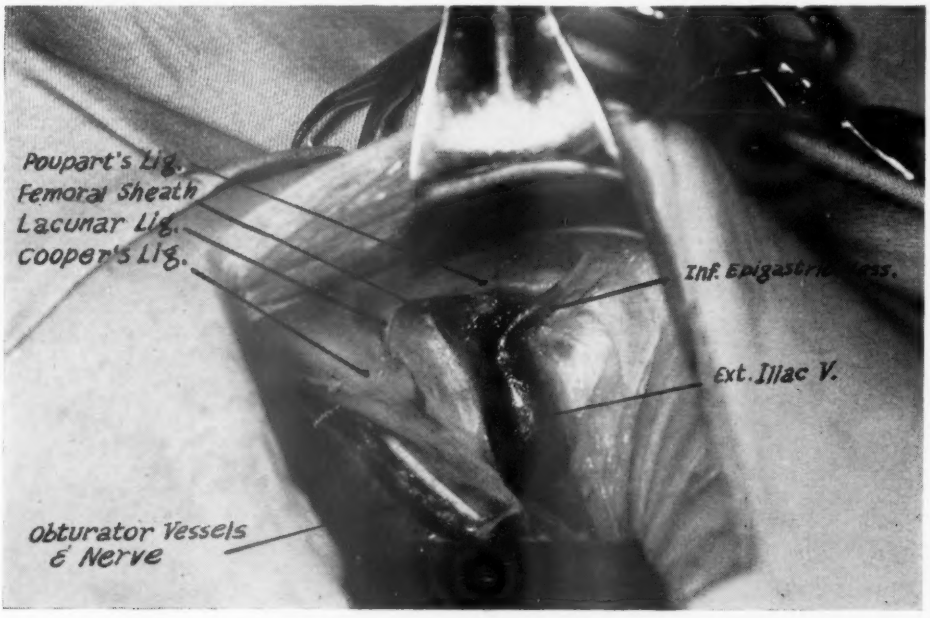

Fig. 3.—Retraction of the peritoneum superiorly and posteriorly widely exposes the femoral area. In actual practice, at this point the hernia sac is teased from the femoral ring. An accessory incision either inguinal or femoral has never been required. The close proximity of the obturator vessels and nerve is demonstrated.

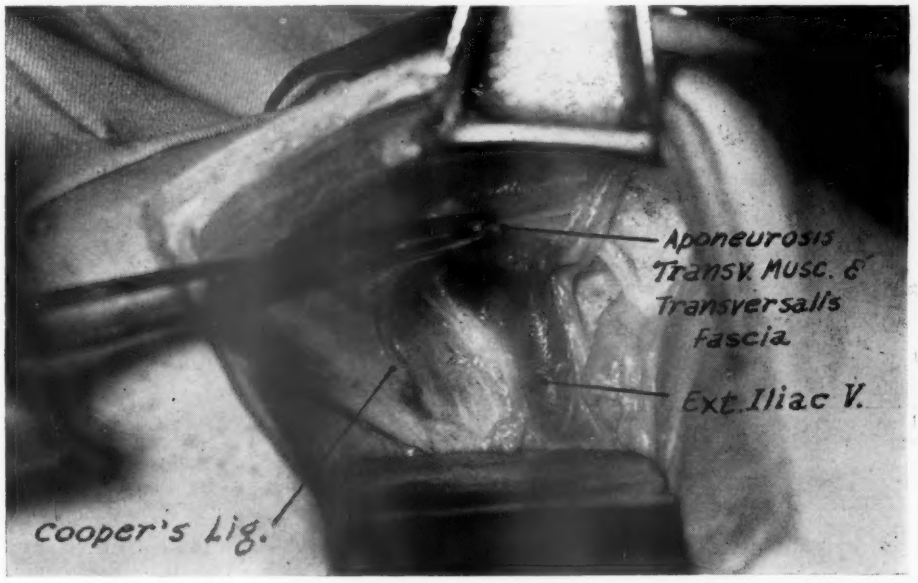

Fig. 4.—The fold of fused transversalis fascia and aponeurosis of the transversus muscle is grasped by a thumb forceps. It appears that the forceps are grasping Poupart's ligament, but actually they have grasped the fused transversalis fascia and aponeurosis of transversus muscle 1½ to 2 cm. above Poupart's ligament and are tenting them forward.

use should not be limited to bilateral hernias. In all patients with unilateral femoral hernia, the opposite side is explored. This additional exploration requires no more than two or three minutes' additional time and does not increase morbidity. In several instances a frank unsuspected hernia has been found

Fig. 5.—The fold of fused transversalis fascia and aponeurosis of transversus muscle is brought down to Cooper's ligament, thus obliterating the femoral ring and lying in juxtaposition to the external iliac vein.

Fig. 6.—The most lateral suture has been placed, and the ease with which the hernioplasty is accomplished is shown by the placement of the second suture.

on the opposite side. More commonly, an enlarged femoral ring is encountered, which can be quickly and easily repaired if desired.

This approach is especially useful for strangulated hernias. In such instances the incarcerated bowel is released in the usual fashion by incising

the lacunar ligament or Poupart's ligament if necessary. If resection or further exploration of the bowel is necessary, the peritoneal incision is extended superiorly as far as desired so as to result in a routine lower abdominal laparotomy incision.

Further advantages are apparent. An anomalous obturator vessel is readily recognized and dealt with. The possibility of injury to the external iliac vein and urinary bladder is minimized. A bilateral femoral hernioplasty can be accomplished in conjunction with any lower abdominal operation. While obesity has been cited as a contraindication,[4, 5] we believe that this factor interferes no more in this approach than it does in any abdominal operation, and we have employed this approach in several obese patients. An associated obturator hernia, one of which was encountered in this series, is readily accessible.

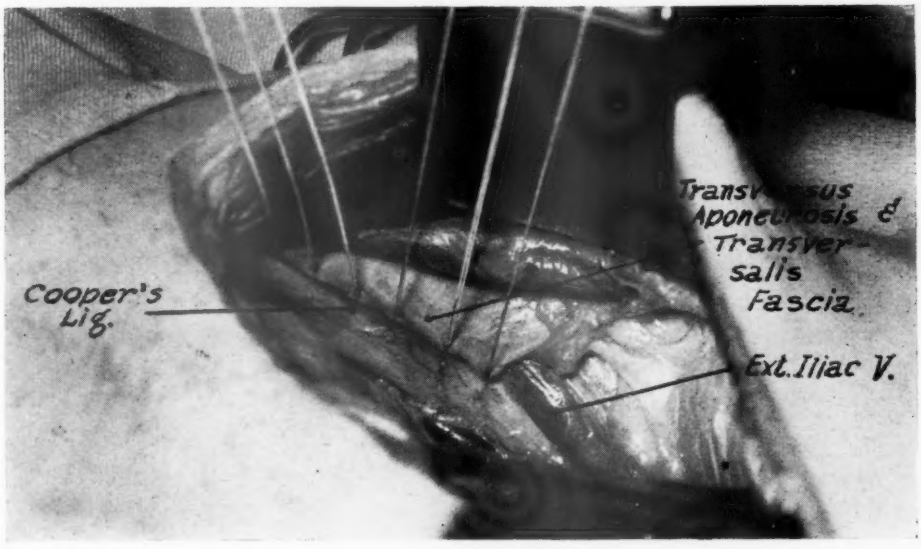

Fig. 7.—The completed hernioplasty.

While we are not here reporting or necessarily advocating the use of this approach for inguinal hernias, several such bilateral hernias, both direct and indirect, have been so repaired. In the case of the direct and large indirect inguinal hernias, a type of repair essentially the same as that which we employ for femoral hernias is carried out. In small indirect inguinal hernias, the sac is excised and the size of the internal inguinal ring is reduced by placing one or two sutures through the fused transversalis fascia and aponeurosis of the transversus muscle on the medial aspect of the ring.

From the accompanying illustrations it is seen that the femoral hernioplasty is accomplished by suturing a fold of the fused transversalis fascia and aponeurosis of the transversus muscle to Cooper's ligament. This results in a strong two-layer repair. The lateral edge of this structure lies in juxtaposition to the external iliac vein and does not result in compression of the vein such as may occur when Poupart's ligament is sutured to Cooper's ligament.

The use of a fold of this structure in place of an incised flap not only doubles the strength of the repair, but obviates a lateral triangular defect such as may occur in the routine McVay hernioplasty.

SUMMARY

Emphasis is directed to the advantages of the suprapubic extraperitoneal approach for the surgical correction of femoral hernia. A modification in the technique of the actual hernioplasty is presented. One hundred thirteen patients with femoral hernias have been successfully operated upon in this fashion at the Los Angeles County Hospital with no known recurrences.

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THE TREATMENT OF INFECTION AFTER MEDULLARY NAILING

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THE introduction of noncorrosive metals and the use of antibiotic drugs before and after operation render the fixation of the fragments of the fractured shaft of a long bone by means of a long heavy medullary nail a relatively safe and practical procedure. Consequently, this method of treating fractures is becoming increasingly popular. Its popularity is justified because the method lessens the periods of hospitalization and of disability, and in skillful hands yields better results than do more conservative methods.

However, treatment of major fractures by intramedullary nailing is fraught with complications, and one of the principal dangers is infection.

It is our impression that postoperative infection occurs more frequently after medullary nailing than it does after other major surgical operations. There are several reasons for this: (1) Wounds of the buttock and lower extremities are more likely to become infected than are similar ones elsewhere. (2) The skin over the operated area may be in poor condition, especially if the extremity has been in traction or in a cast for some weeks, or pressure sores may be present. (3) The tissues have been traumatized and some of them are devitalized, and the wound may contain pieces of devitalized bone. (4) The operation may be quite traumatic, and this is especially true if excessive shortening has occurred or if the operation is performed by a surgeon who is not skilled in this procedure. (5) A large foreign body is left in the wound. (6) The patient may be anemic and debilitated from hemorrhage, shock, or prolonged rest in bed, or he may be suffering from some other disease, such as malignancy, and the operation may be undertaken as a lifesaving or palliative measure. (7) The fracture may be a fresh compound fracture or one in which the wound has been closed, and may have been contaminated or even infected, or it may have been mildly infected by a previous unsatisfactory operation for open reduction with or without internal fixation. (8) Hematomas are especially likely to develop in the wound after the insertion of a medullary nail, and these may become infected. (9) A nail which is too long or one which has migrated may erode through the skin and thus become infected.

The infection may occur at the site of the fracture or near the end of the bone in the wound where the nail was introduced and may remain localized in the soft tissues or bone, or it may involve the bone throughout the track of the nail.

During the past few years we have observed a number of patients with infection following the insertion of a medullary nail, and while the majority of these were not seen by us until the bone was chronically infected, in some the nailing was carried out in our clinic and the chronic bone infection developed while the patient was under our care (Fig. 1). In each instance there were some

Received for publication, Aug. 28, 1953.

A. *B.* *C.* *D.*

Fig. 1.—*A*, Closed fracture of both bones of the leg treated elsewhere by reduction and inadequate plating, plus the removal of a large piece of bone. X-ray appearance on admission; wound healed and apparently clean one week after operation. *B*, Following removal of the plate and insertion of a Lottes nail, infection developed. The wound was widely opened and the tube left in depth of the wound for instillation of penicillin. *C*, Two months later chronic infection present, nail still in place, and fracture healing. *D*, Because of persistent drainage, nail and sequestra removed. Fracture united but precarious, wound healed. The bone gradually strengthened and the patient now has full use of the leg and no drainage.

extenuating circumstances, but the result is just as bad as if there were no excuses. From this experience we have formulated some ideas concerning the treatment of infection after medullary nailing which we wish to communicate in this paper. We offer as evidence illustrations of three of the severe infections with chronic osteomyelitis.

DIAGNOSIS OF POSTOPERATIVE INFECTION

It is important that the presence of infection in an operative wound be diagnosed as soon after its onset as possible. This is because the optimum time to treat the infection is early in the process and before the bone becomes involved. Early diagnosis requires vigilance on the part of those in charge of the patient after the operation.

It is usual for the surgeon to think of fever as the first sign of infection, and certainly this is usually one of the earliest signs. However, it is not a reliable sign after medullary nailing because in most of these patients the temperature is elevated for from twenty-four to forty-eight hours after the operation. Consequently, we inspect the wound in such patients, but do not open it unless there are other signs of infection. We are suspicious of infection if the fever lasts more than forty-eight hours or if it develops after the temperature has been normal a few days. Furthermore, absence of fever is not a dependable sign that infection is not present, particularly if the patient has had antibiotic therapy.

When such a patient complains of unusual pain in the wound and especially of a throbbing pain, the surgeon should become suspicious of infection. Other symptoms are headache and general malaise. Increased pulse rate and elevation of temperature may be signs of infection, and this is more likely to be true if increase in the pulse is proportionately greater than the increase in temperature. Corroborative evidence of infection is increase in the leukocyte count, and this is more significant if there is a pronounced increase in young forms of leukocytes in the differential count (shift to the left).

The onset of infection may be ushered in by a chill with high fever, and the patient may become acutely ill within a few hours and the infection pursue a stormy course. More frequently, however, the onset is insidious, and deep infection may be present for several days and the area around the fracture be converted into an abscess cavity before there are sufficient signs and symptoms to justify opening the wound. This is especially likely to occur in patients who have been treated with antibiotics after the operation.

TREATMENT OF INFECTION

Any or all of the symptoms or signs mentioned previously demand that the dressing be removed and the wound inspected. If the tissues are red, swollen, and edematous and the skin exhibits increased heat and is unusually tender, one or two of the skin, and if necessary some of the deeper, sutures should be removed and an instrument introduced into the wound. If a hematoma is encountered and this is not large and clotted, it should be evacuated and the wound edges strapped together with adhesive and a pressure dressing applied.

If a small amount of pus is encountered in the superficial tissues (stitch abscess or small hematoma) and the surgeon is reasonably sure that the infection does not involve the deeper tissues, this should be evacuated and the area left open and covered with grease gauze and a dry dressing or a wet dressing.

If the hematoma is large and the wound contains considerable clotted blood, or if evidence of infection in the deeper tissues is encountered, it is advisable to take the patient to the operating room, administer a general anesthetic, and open the wound under aseptic conditions. There the wound is opened widely and irrigated thoroughly, and if a large hematoma is found it is evacuated and if there is no evidence of infection the wound is sutured. If a bleeding vessel is found it is ligated. If there is considerable bleeding from the bone or if there is much general oozing, a rubber tissue drain should be left in the wound and a moderate pressure dressing applied.

If there is evidence of infection, any loose fragments of bone or bone grafts which may be in the wound are removed, devitalized soft tissues are excised, and the wound is again irrigated thoroughly. This is because the loose bone fragments and grafts are not viable and, if left, will act as irritating foreign bodies which hinder healing, harbor infection, and will have to be removed later. The nail is not removed, and if it is found that it is not fixing the fragments firmly, the limb should be immobilized, preferably in a large plaster cast. Then the wound may be packed open loosely with petrolatum gauze, but we prefer to place one or two rubber tubes in the depths of the wound and pack it loosely with gauze soaked in normal salt solution (Fig. 1). Then a massive hot wet dressing as applied and a solution of penicillin is instilled into the wound every three hours, or it may be irrigated continuously with a penicillin solution. The solution used is 1,000 units of penicillin per cubic centimeter of normal salt solution. The dressing and pack are changed in twenty-four to forty-eight hours, and after seven or eight days, if the wound is clean it may be closed by secondary or delayed primary suture.

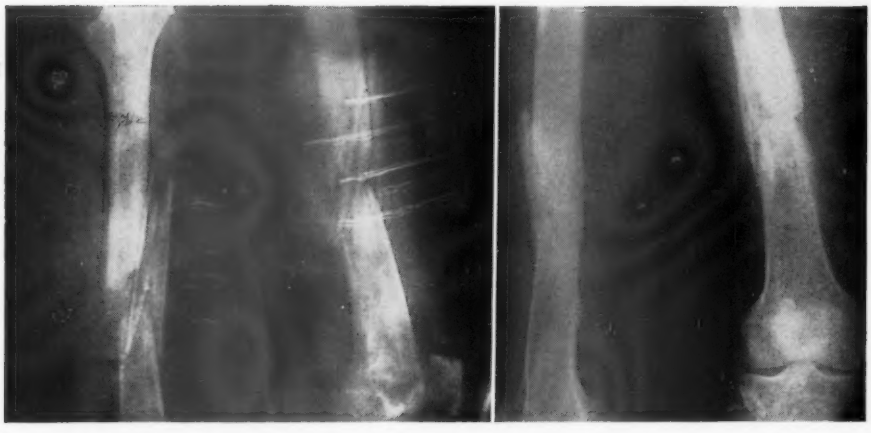
Cultures of the wound are taken when it is opened, and the organisms are tested for sensitivity to antibiotics and the appropriate drug (penicillin, streptomycin, Aureomycin, Terramycin, and so forth) is used systemically in full dosage. Until the laboratory findings are reported, the patient should be given penicillin and some broad spectrum antibiotic (streptomycin) in full dosage.

If the infection does not subside within a few days to a point where it is considered safe to suture the wound, it is probable that the bone is involved and it is possible that the infection may have entered the marrow canal and extended upward or downward along the track of the nail. In such a case the nail should not be removed until union is present, but antibiotic therapy should be continued, and the wound may be explored again for loose pieces of bone and then packed open with petrolatum gauze while the limb is immobilized, preferably in a large plaster cast.

There is still a possibility that the infection may subside, the wound heal, and the bone unite without sequestration. The wound is dressed infrequently and the petrolatum gauze pack changed at each dressing. It is thus encouraged

to heal by granulation. During this period the fragments and the extremity are immobilized in a cast or splint, and the fracture has an excellent opportunity to heal. Usually under such treatment the fracture does heal unless there is diastasis or sequestra are present.

A. *B.* *C.*



D. *E.*

Fig. 2.—*A*, X-ray appearance on admission three and one-half months following nailing of an open fracture and wound closure as a primary single-stage operation. Débridement and drainage of the wound failed to improve its condition. The large central fragment was a sequestrum. At a second operation it was removed and the fragments pushed together with the nail still in place.

B, Following sequestrectomy the wound continued to be unfit for closure. The nail loosened and began to back out and had to be removed. The wound and medullary canal were irrigated by constant drip of penicillin with three catheters in the canal (1,000,000 units to the liter). Closure was performed two weeks later. This was complicated by a postoperative hemorrhage that required opening this wound and ligation of a vessel, followed three days later by a secondary closure. The wound healed and remained healed.

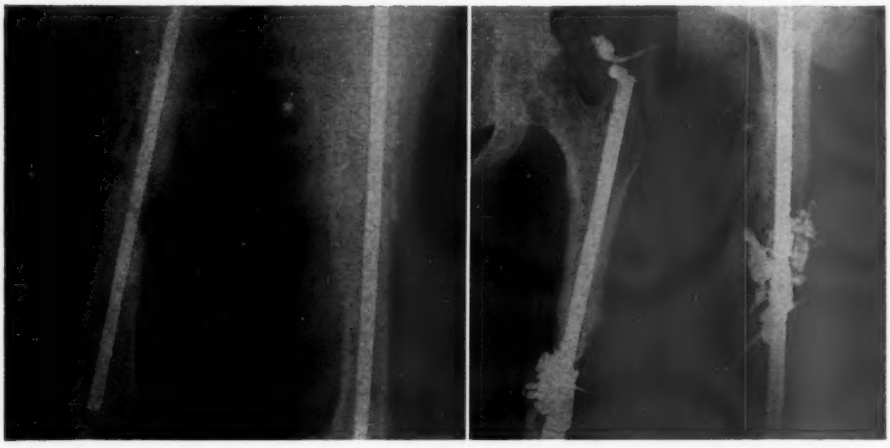
C, Appearance after a period of three months of immobilization in a cast. Wound healed.

D, Because of non-union, autogenous bone grafts used to bridge the defect.

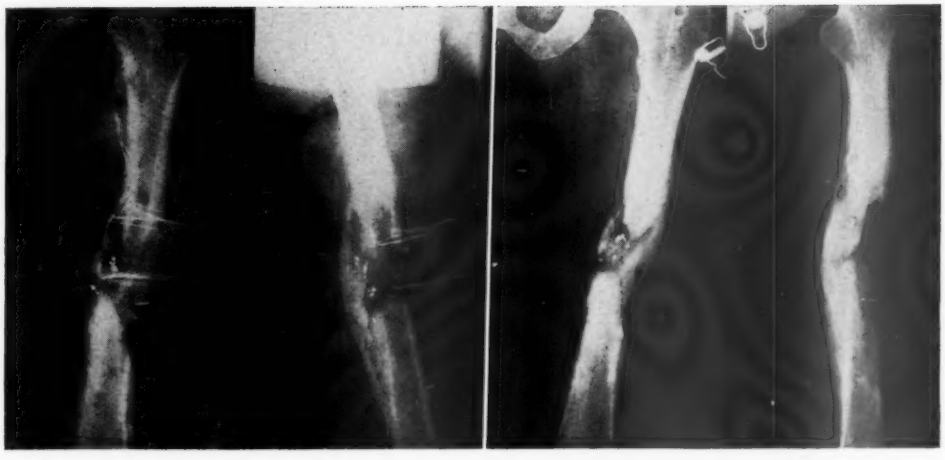
E, Present condition of the femur: wound healed, fracture healed, but there remains considerable stiffness of the knee.

Sometimes after prolonged infection the end of one or of each fragment may become necrotic and be sequestrated as ring or cone-shaped sequestra. These and other sequestra should be removed soon after they are discovered, and the

bone ends should be forced together and immobilization and free drainage continued. This is true even though the sequestrum is large and comprises an entire section of the shaft. Such sections, like ring sequestra, should be split into two



A. *B.*



C. *D.*

Fig. 3.—*A*, Appearance on admission fourteen months following a closed fracture of the femoral shaft which was treated by open reduction, plating, and cast. After four months another operation was performed, the plate removed, an intramedullary nail inserted, and iliac graft implanted. Postoperative infection developed.

B, Injection of sinus tract with Lipiodol revealed extensive involvement of medullary canal. Nail was removed and canal irrigated with penicillin while wound was packed open with three catheters in the canal, immobilized in a plaster spica cast.

C, Appearance following removal of the nail; beginning of union.

D, Wound healed, union still precarious but patient walking without support. Improvement has continued.

or more sections and removed from around the nail (Fig. 2). We believe that in such instances it is good surgical judgment to accept a moderate amount of shortening rather than court a chronic infection and non-union.

Occasionally infection extends up and down the medullary canal along the entire track of the nail. In such instances it is advisable to remove the nail whether union is present or not and to remove any dead bone and infected granulation tissue which can be identified, and then the medullary canal can be irrigated and even scrubbed by running strips of gauze from the trochanter down to the fracture site, and the canal in the distal fragment can be curetted lightly and irrigated with a catheter (Fig. 3). Catheters, usually three (one in either end of the proximal fragment and one in the distal fragment), are left in the wound, and penicillin solution (1,000 units per cubic centimeter) is either instilled into the catheters every three hours or is administered by a continuous drip. The wounds are packed loosely with petrolatum gauze, and the limb is immobilized in a large plaster cast, the catheters being led out through the cast. In ten days or so it may be possible to close one or both wounds by a secondary suture (Fig. 2). If this does not seem possible, they may be permitted to heal by granulation tissue (Fig. 3).

In addition to the chronic disease of the bone and non-union of the fracture, the infected case of medullary nailing may be further complicated by infection and eventual stiffening and ankylosis of the joints above and below the fracture. This is especially likely to occur if the nail enters the joints.

In every instance where infection has occurred after a medullary nailing, the general health of the patient is a primary consideration, and he should be given supporting treatment: transfusions of whole blood, high-calorie and high-protein diet, intravenous feedings if necessary, excess vitamins, the most judicious antibiotic therapy, and careful and assiduous nursing care. At the same time, the patient should be made as comfortable as possible and pain relieved by proper attention to the wound and immobilization of the fracture, rather than by drugs.

If the fracture does not heal, autogenous bone grafts may be placed across the fracture site in order to expedite union. This is done after the infection has subsided and the wound has been closed or has healed by secondary intention. With the protection afforded by antibiotics, the bone grafting operation need not be delayed six months or more, as in the past, but can be carried out within a few weeks after the wound is healed (Fig. 2).

In exceptional cases where it seems unlikely that a useful extremity will be obtained even after further prolonged treatment, amputation is the procedure of choice. Decision to amputate the extremity is favored by advanced age of the patient, poor general condition of the patient, and failure to improve this by transfusions, high-protein diet, relief of pain, and other conservative measures, inability to eradicate the infection, failure to obtain union, excessive shortening of the extremity, damage to the nerve or blood supply of the extremity, and inability of the patient to finance prolonged hospitalization and indefinite disability. This last should not be a factor, but it cannot be ignored.

PREVENTION OF INFECTION

When one considers the difficulty which is sometimes encountered in the early diagnosis of a deep postoperative infection and the dire results which

may follow if the bone becomes involved, it is evident that even without the use of antibiotics special care should be taken to avoid infection after medullary nailing. This demands not only that the procedure be performed only by qualified surgeons who are experienced in the open treatment of fractures, but that such surgeons take the care to have on hand the proper equipment before the operation is started. During the operation the tissues should be handled gently, extensive stripping of the periosteum avoided, and the nail inserted and the reduction effected with as little trauma as possible. Also, only those cases in which the fracture is suitable for this type of fixation should be treated with a medullary nail; that is, they should be simple transverse or short oblique fractures of the shaft without too much comminution and not too near the end of the bone. Likewise, some care should be exercised in selecting the patient and in preparing him for the operation. If he is in shock or impending shock the operation should be delayed until he has reacted satisfactorily. If he is debilitated or the skin is in poor condition, the operation should be postponed until these are corrected, unless fixation of the fracture is considered to be so important that the nail may be inserted as a lifesaving procedure.

In compound fractures it is a moot question whether or not the wound should be débrided and the nail inserted at the primary operation and the wound either closed or left open to be closed later by delayed primary or secondary suture. Débridement, insertion of a medullary nail, and primary suture of the wound have been carried out successfully in many instances where the tissue damage was not extensive and the compound fracture had had antibiotic therapy, and was operated upon within less than twelve hours after the injury.

However, in a compound fracture, prevention of infection and closure of the wound are the primary aims of treatment, and treatment of the fracture is secondary. With full realization of the benefit afforded by immobilization in preventing and combating infection, in promoting healing of the soft tissues and of the bone, and in adding to the comfort of the patient, we believe that in most instances internal fixation should not be applied at the primary operation, but the wound should be débrided, and after it has been closed by primary, delayed primary, or secondary suture or by secondary healing, then the medullary nail may be inserted or other internal fixation applied if indicated. In the meantime, the fracture should be immobilized by external fixation (traction or a cast, or a splint).

If postoperative infection occurs in a compound fracture after medullary nailing, the condition is treated just as described previously. We have had no experience with inserting medullary nails in the presence of frank infection in the bone; however, this has been carried out with a fair degree of success by others.[1, 2]

DISCUSSION AND SUMMARY

It is emphasized that infection is especially likely to occur after insertion of a medullary nail and that this may lead to prolonged illness and disability and even to loss of life or the limb. Consequently, the patient should be watched carefully after the operation, and at the first signs of infection the wound should

be inspected and, if necessary, the wound opened widely, and loose bone, but not the nail, removed and vigorous treatment of the infection instituted. Such early treatment may abort or eradicate the infection before the bone is involved and permit the fracture to heal in the usual manner.

If the bone becomes infected, sequestra should be removed when discovered, but the nail should be left in place until union occurs because the immobilization which it affords is a valuable asset in limiting spread of the infection and promoting healing of the fracture. In such cases the treatment is similar to that now used in the treatment of chronic osteomyelitis. If the infection spreads up and down the track of the nail and this becomes loose and inefficient, it should be removed and the fracture treated as an infected compound fracture with or without union.

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THE TRACTION BRACE

A New Traction Method in the Conservative Treatment of Intervertebral Disc Lesions

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THE present concept of intervertebral disc lesions is built on the pathologic data, the operative findings, and the clinical picture which have been described in the literature during the last fifteen to twenty years. The ruptured or herniated intervertebral disc is recognized generally as a common cause of low-back disability.

Speed[1] found that 80 per cent of patients who complain of low-back pain combined with true sciatic radiation are regarded as having disc injury with probable protrusion. The syndrome occurs most commonly in young men who make their living by doing hard manual labor, such as farmers, ranchers, mechanics, and so forth. Sell[2] reported a series of 252 patients, in which 25 per cent eventually had to be operated upon.

As a practical plan, the previously mentioned authors[1, 2] recommended conservative treatment of patients who have a probable herniated disc.

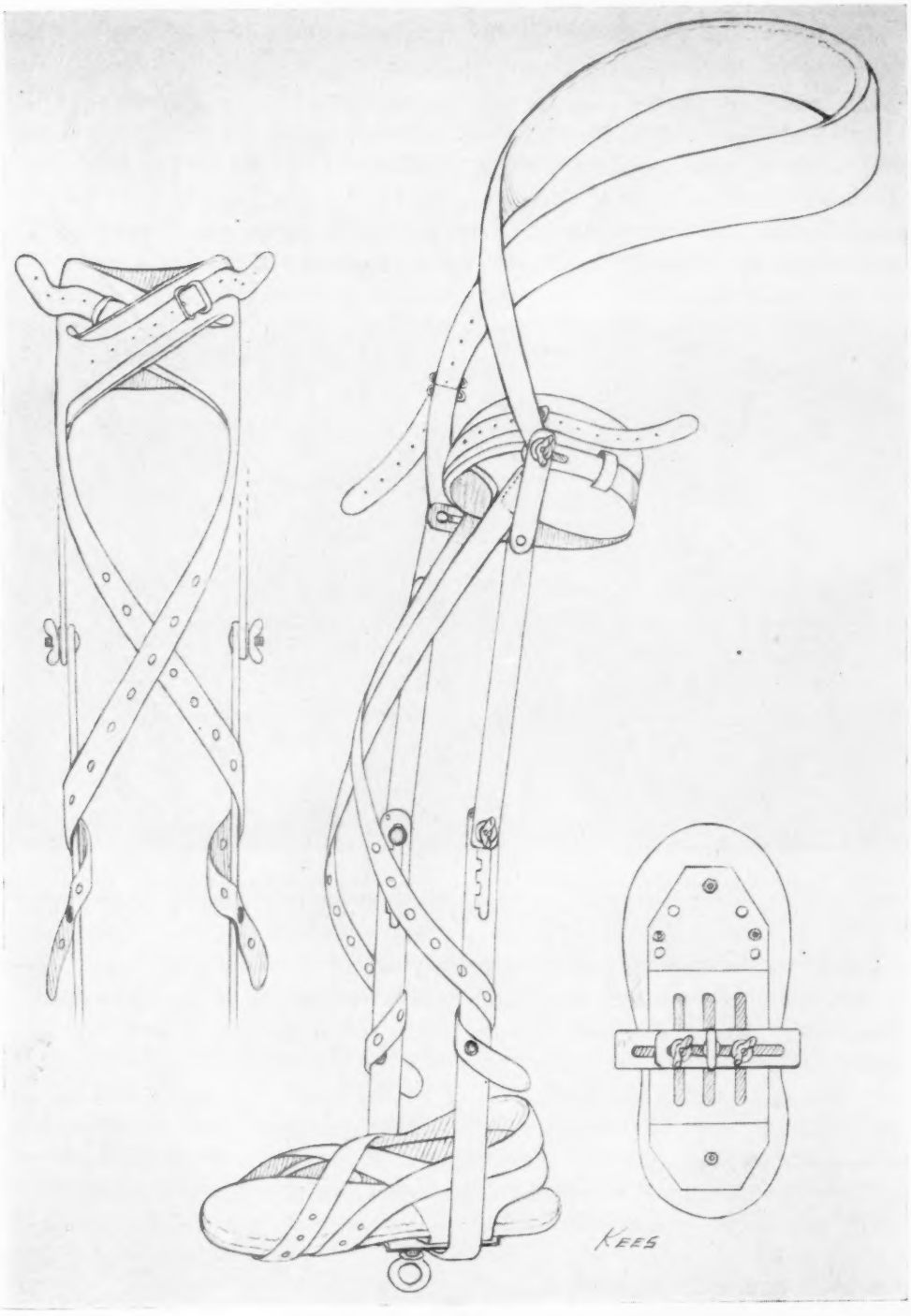
The majority of patients who present the syndrome of a ruptured disc may be relieved temporarily or permanently by nonsurgical measures.

The conservative treatment consists of: (1) Complete rest on a hard bed with boards under the mattress; (2) heat diathermy (only occasionally); (3) antispasmodics, such as Tolserol (mephenesin); (4) traction for a duration of ten to twenty days, with slight flexion of the bed under the knee; (5) plaster jacket or low-back brace, in some severe cases after the traction.

The most important of all these means is the traction. It is carried out by so-called skin traction with five to ten pounds on each leg. The legs are shaved, washed with soap, and painted with tincture of benzoin. Then the skin traction is applied by means of skin plaster tapes and elastic bandages. This method of traction has the disadvantages that the plaster straps are torn frequently and the traction has to be reapplied. Furthermore, it is frequently complicated with irritation of the skin, swelling of the ankles, and, in a number of cases, allergic reaction to the plaster tapes. Dermatitis with blisters and painful sensation results, and the traction has to be removed. Instead of the specific treatment, there is treatment of dermatitis for a few days or weeks. This is time consuming and an unnecessary expense for the patient; it means loss of time from work and extended hospitalization. A large number of patients develop dermatitis and pain in the legs resulting from the skin traction. Some of the diabetic or allergic patients get very severe dermatitis. It appears on the next day approximately twenty-four to thirty-six hours after the application of the plaster tapes. It starts with itching and pain under the tapes followed by redness and swelling. The patient is very uncomfortable and asks for immediate

Received for publication, Aug. 28, 1953.

removal of the traction. When the tapes are removed, there are large areas of redness of the skin and large blister formations measuring approximately 3 to 4 cm. in diameter. Some of the blisters rupture leaving superficial ulcerations.



A. *B.* *C.*

Fig. 1.—Drawing of the traction brace. *A*, Front view of lateral adjustable bars and knee leather lacings. *B*, Lateral view of the traction brace as a unit. The crossed leather straps prevent slipping. *C*, Bottom view of the foot plate.

The inflammation subsides in a few days after the removal of the plaster tapes, but several days or weeks are necessary until the ulcerations heal. During this time no traction can be applied. Also, these patients develop dermatitis when the skin traction is reapplied, and therefore an adequate treatment for them is not possible.

Having in mind these defects, a brace was designed and perfected. The traction brace* is made from a light metal (aluminum), and is adjustable in length and width. This is made possible by using perforations and movable screws on the lateral bars, semicircular band, and foot plate. The foot plate is made from metal and wood, and felt-lined with foam rubber. There is a permanent knee joint which permits movement in the anterior-posterior position. The brace can be adjusted to many different contours of the lower extremities.

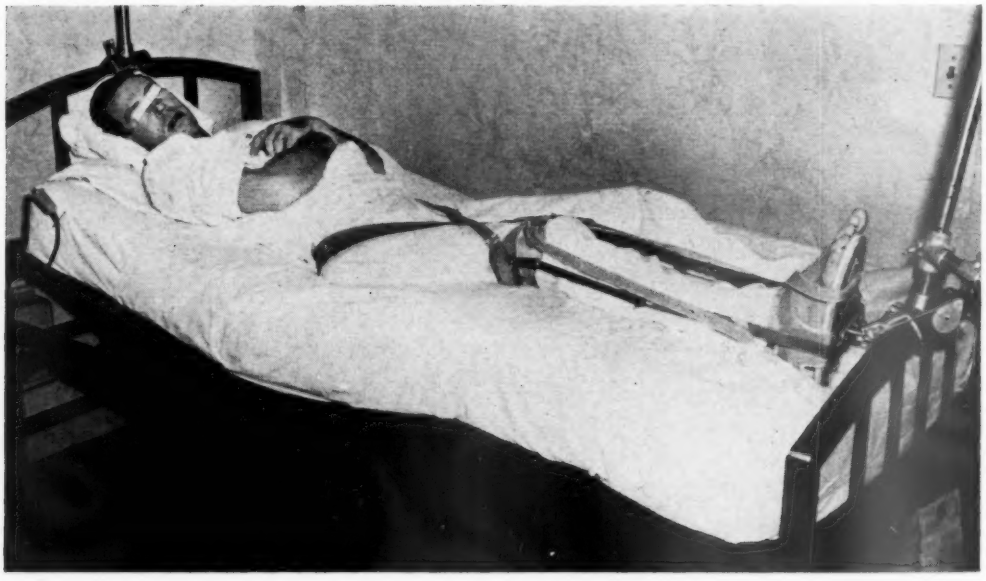


Fig. 2.—Photograph of the traction brace in use. The straps are felt-lined by foam rubber and attached to the hip, knee, and foot. It is used with five, ten, or fifteen pounds of weight. The knee is slightly flexed by breaking the bed at that level.

Leather cuffs and straps are attached to the metallic part of the brace. They are felt-lined with cemented foam rubber so that slipping and compression of the vessels and nerves are avoided. The leather cuffs over the knee are attached posteriorly on the semicircular band; they cross each other anteriorly and posteriorly around the lower portion of the thigh to prevent any slipping. They are made as tight as desired by means of buckles. Two leather straps extend down on both sides of the knee and cross over the anterior aspect of the lower leg. They are attached to the lateral bars, using small buttons, one on each side. The leather cuffs above the knee are to prevent slipping by the patella, and therefore the knee has to be slightly flexed using a pillow or breaking the bed under the knees slightly.

Two leather straps on each side of the foot cross around the heel and the dorsum of the foot and are attached to the foot plate by buttons. They hold the

*Manufactured by Max Wocher & Son Co., Cincinnati, Ohio.

foot firmly in place. The heel itself is free and elevated from the bed so that no pain or discomfort occurs. The foot plate is adjustable to the lateral bars to keep the heel free and to give the best comfort.

A leather strap fixed permanently on the upper end of one of the lateral bars goes around the hip for traction in intervertebral disc lesions and muscular spasm, or around the middle of the thigh for traction in upper femur fractures and postoperatively in hip prosthesis. It crosses the anterior aspect of the upper thigh and is attached on the opposite lateral bar by means of a buckle. These straps are also felt-lined with foam rubber cemented to the leather (Figs. 1 and 2).

The process of applying the brace is best carried out in a hospital, where supervision and care of the patient are best given. It can be used also at home by attaching any kind of weight in any way over the bed and using a pillow under the knee for slight flexion of the knee. The application is so simple that an intelligent patient will be able to do it himself after instruction by the physician. The brace as a whole may be adjusted during the application upward or downward in length and laterally in width to permit the most comfortable position for each leg. It can be used with five, ten, or fifteen pounds of weight on either the left or right leg. The time required is minimal; two or three minutes is sufficient for application as compared to fifteen or twenty minutes for the skin traction.

The orthopedic and neurosurgeons will find the traction brace especially useful in the conservative treatment of intervertebral disc lesions. It has been used with great satisfaction. The brace is one of the simplest and most variable devices. It can be used for a large number of conditions, both as an adjustable splint and as a traction brace for partial immobilization and traction on the lower extremities.

SUMMARY

1. The skin traction method is complicated by dermatitis and is time consuming.

2. A new traction brace has been described for conservative treatment of intervertebral disc lesions. The brace is made from aluminum and has a foot plate of wood, felt-lined with foam rubber. It is attached to the lower extremities by means of leather lacings and straps felt-lined with foam rubber.

3. The traction brace is adjustable, simple, and rapid to apply and gives the most comfortable and smooth traction for hospital and home use with five, ten, or fifteen pounds of weight.

4. The traction brace can be used in all conditions where traction on the lower extremities is required, such as low-back disability, pain from muscular spasm, upper femur fractures, and postoperatively in hip prosthesis.

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PROMINENT COSTAL CARTILAGES (TIETZE'S SYNDROME)

WILLIAM C. BECK, M.D., AND SAMUEL BERKHEISER, M.D., SAYRE, PA.

TIETZE, in 1921,[1] reported four cases of a peculiar syndrome for which he had no explanation. He described it as "swelling of costal cartilages of unknown etiology, insidious in onset, spontaneously painful, not associated with constitutional disturbances and of a prolonged fluctuating course." Since this time, there have been sporadic reports of similar cases and, in some instances, rather large series. These have been reported in the European, the British, the Canadian, and the Japanese literature. Not until June of this year, however, has a report of similar cases been seen in the American literature. In Dean Lewis' *Practice of Surgery*,[2] in the chapter on afflictions of the thoracic cage, Heuer and his associates showed two pictures which unquestionably described a similar condition. A letter from the publisher states that these photographs appeared in the original edition of Lewis' *Practice of Surgery* which was published in 1930, but that the photographs were received with the text for Chapter 5 on Oct. 6, 1927. There is no doubt that American surgeons have observed similar cases. In the last two years, we have seen four such instances which lead to this report. From our experiences with these cases, we are not certain that this is a disease of the costal cartilage at all.

Judging from published reports, there is no certainty as to the etiology, or even any exact definition of the pathology, of this condition. Only in their clinical symptomatology do these patients fall into a group.

Tietze described the syndrome as a cartilage dystrophy, probably due to wartime malnutrition. In one of his patients who was subjected to biopsy, there was no evidence of inflammatory change. Gill, Jones, and Pollak[3] reported five cases, in one of which a biopsy was performed. In none of these was malnutrition a factor. In Geddes'[4] series of thirty cases, all of which occurred in Canadian soldiers, there was no evidence of any malnutrition. Some authors have noted an associated respiratory tract infection. In many other cases, however, this was not present.

The clinical signs of the syndrome are consistently uniform. In most of the reported cases, as well as in ours, a healthy, usually young adult of either sex reports to the physician because of a painful swelling situated over one of the costal cartilages. The most frequently affected cartilage is the second, more often on the left than on the right. Less frequently, the third left costal cartilage or, rarely, one of the other costal cartilages is involved. The lesion is usually limited to one costal cartilage but in rare cases has been reported simultaneously present in two or three. There is a minimal amount of pain, usually described as a dull aching pain deep in the chest beneath the prominent cartilage. The pain appears to precede the appearance of the cartilaginous swelling or protrusion. It is rarely severe enough even to require the mildest of anodynes. The

Received for publication, Aug. 31, 1953.

pain is fluctuant in its course and may disappear, only to reappear after a time. The patient and, indeed, the surgeon often fear that this might represent a tumor of some sort. There are no constitutional symptoms.

In almost all series reported, occasional biopsies have been taken. In Geddes' rather large series of thirty cases, one patient was subjected to biopsy. Rather surprisingly, in none of the reported cases have we found that the entire cartilage was removed. Of the biopsies which are reported, one can only say that they all apparently have had the appearance of normal costal cartilage, and in none was there any evidence of tumor formation. In some of the reports there is mention of hyperplasia of the cartilage and occasionally mention of calcification or bone formation. In our opinion these reports are consistent with the finding of normal cartilage.

We must admit that until the article by Motulsky and Rohn[5] appeared we had seen only the report in Lewis' *Practice of Surgery,* most of the previous articles in the literature having been buried under the terminology of Tietze's syndrome. Therefore, in the four cases reported herein, the rib cartilage was removed. This has resulted in our acceptance of a different concept of the syndrome than is general.

The second costal cartilage was involved in all four of our cases. The clinical history was identical with reports in the literature: a painful swelling appearing at variable times in the region of the second costal cartilage.

Physical examination revealed a marked and definite prominence at this point, which was mildly tender to palpation and to percussion. There were no constitutional symptoms. The x-ray examination was entirely negative in all of the patients. At operation, the cartilage was found to be approximately normal in size and thickness. It was, however, buckled forward in an acute angle. Immediately subadjacent to the cartilage, heavy ligamentous fibrous tissue bands were visible. These were neither incised nor resected. Histologic study of the removed cartilages proved them to be entirely normal.

It appears to us, therefore, that the disease is not specifically one of the costal cartilage, but that some other process causes the buckling. It is interesting to note that in the one patient in Tietze's series who was subjected to surgical intervention, the surgeon described the cartilage as being "buckled forward" (vorgebückelt).

It is noteworthy that this condition affects the second costal cartilage oftener than any other. In the complete absence of any occupational or traumatic history in the reported cases, one must search for an anatomic reason for this peculiar localization. There is nothing in the bony blood supply or in the histologic anatomy of the second cartilage which differentiates it from any other of these cartilages. The only difference appears to be in the ligamentous fibers which surround the costal cartilage. According to Spalteholz,[6] a fibrocartilaginous layer, the interarticular chondrosternal ligament, extends from the medial end of the second cartilage to the cartilaginous or bony junction of the manubrium with the body of the sternum. This constantly divides the chondrosternal joint into two separate joint cavities. The other cartilages have somewhat similar

fibrous strands, but these strands are different from the second. They lead generally to only incomplete division of the joint cavity. With such an intricate ligamentous arrangement, it is quite probable that in certain anomalous situations, a similar arrangement might take place behind the third or fourth costal cartilage. From these findings we would suggest that the disease entity described by Tietze may not be one of the cartilage at all. There may be an inflammatory reaction contracting the ligaments lying immediately behind the cartilage, producing the consequent buckling.

The therapy of this condition apparently needs little comment. The other inflammatory and noninflammatory diseases of costal cartilage need to be differentiated. If it is certain that one is dealing with such a buckling of cartilage, then reassurance of the patient is probably all that is needed. We have resected the cartilage in our patients with a cure of symptoms in all. When the cartilage is resected, we have followed the suggestion of Maier[7] to leave a cuff of perichondrium to insure healing. We have removed the cartilage and assured ourselves of a cuff of perichondrium by curetting a small portion of the cartilage away on either side.

CASE REPORTS

CASE 1.—A 22-year-old unmarried woman presented herself with a lump over the second costal cartilage which had been present for approximately four or five years. She felt that it had grown slowly during this time, especially recently, and had in recent weeks prior to admission noticed mild pain in this area. At the onset of the condition years previously, she had also noted some pain beneath the sternum. Other than the protruding mass over the second costal cartilage, the physical examination was entirely negative, with the exception of a small lump present in the left breast. Under general anesthesia, the second costal cartilage was resected and found to be buckled forward. A fibroadenoma of the left breast was also removed. The patient made an uneventful recovery. She was seen two years after the removal and had had no recurrence of symptoms.

CASE 2.—A 34-year-old woman had noticed a lump over the left second costal cartilage for approximately three years. It was occasionally painful, especially when she did excessive work, such as ironing. Physical examination revealed a mass of about 2 or 3 cm. in diameter over the second left costal cartilage. Physical examination was otherwise entirely negative. Under general anesthesia, an incision was made over this costal cartilage and it was found to be buckled forward at this point. The cartilage was removed. The patient was seen six months later and found to be entirely free of symptoms.

CASE 3.—This patient complained of a mass over the second right costal cartilage which had existed approximately two months. It was moderately painful at the time of onset, and she had noticed some pain beneath the sternum for approximately two or three weeks before the mass itself was noted. Physical examination revealed a mildly tender mass about 2 cm. in diameter over the second right costal cartilage. Physical examination was, otherwise, entirely negative. Under general anesthesia, an incision was made over the second costal cartilage. The cartilage was found to be buckled forward; it was completely resected. The postoperative course was uneventful. The patient was seen about one year later and stated that there had been no recurrence of symptoms.

CASE 4.—A boy of 11 years was seen because of a mass over the second right costal cartilage of one week's duration. There was no history of trauma. At the onset of this condition, the child had complained of a slight aching in the right chest and arm. The aching disappeared after about twenty-four hours and had not recurred. Physical examination was otherwise completely negative. At exploration, an anomalous bifid rib was found with an

abnormally buckled forward costal cartilage. The cartilage was completely resected and the wound closed. The child was seen two years following surgery, and there has been no recurrence.

SUMMARY AND CONCLUSIONS

A prominence of a costal cartilage had been reported in the literature for some time as a specific syndrome under the name of Tietze's syndrome. This occurs most frequently on the second costal cartilage. We reported four cases in which the costal cartilage has been completely removed. In these we have found that the cartilage itself was not diseased, but appeared to be buckled forward. The anatomic explanation which we would like to suggest is that this is a disease of the ligament which lies subadjacent to the second costal cartilage rather than a disease of the cartilage itself, and that a contracture of this ligament causes a buckling forward of the costal cartilage.

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ACCURACY OF PREOPERATIVE X-RAY DIAGNOSES OF BREAST TUMORS

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ROENTGENOLOGY must impart additive or confirmatory information if it is to attain its rightful place among procedures useful in the preoperative diagnosis of breast tumors. Following recent improvements,[1] it is now possible to make some preliminary assessment of advances afforded by roentgenography of breast tumors. This subject has been static during the past twenty-five years. Dr. George E. Pfahler attempted diagnostic x-ray studies of the breast in 1930 when he successfully demonstrated several cases of cancer to the members of the Medical Literature Club in Philadelphia.[2] He never published his findings since he felt then, as most surgeons do today, that an x-ray examination of the breast could add little to an expert clinical examination. He therefore remained satisfied to use the x-ray examination only when he believed it would help him in a differential diagnosis. Our experience has taught us, however, that the x-ray examination should be used routinely since its disclosures often come as a surprise.

METHOD AND RESULTS

The last 210 consecutive x-ray studies of breast tumors in the Albert Einstein Medical Center (Northern Division) were interpreted by three roentgenologists, twice without and finally with knowledge of the clinical findings. After these readings were analyzed for consistency among themselves, they were compared with the pathologic findings.

Of the lesions in these 210 cases, 163 were benign and 47 were malignant. During the first round of x-ray interpretations, erroneous diagnoses were made in approximately 10 per cent by all three roentgenologists. With each successive review, a slight improvement in accuracy of diagnosis occurred, chiefly because each roentgenologist gained more experience in adhering to the diagnostic criteria previously formulated.[1]

When the third session of interpretations was concluded, erroneous diagnoses had been made in only 5 per cent, chiefly because of better recognition of malignant lesions and because the clinical findings also were taken into consideration (Table I). The inconsistencies of the individual interpretations were surprising, being subject to the "human error" encountered by Garland and associates.[3]

Very encouraging was the high accuracy (approximately 97 per cent) achieved by all roentgenologists in the diagnosis of benign lesions, but the accuracy of x-ray diagnosis of malignant lesions never exceeded approximately

Received for publication, Sept. 8, 1953.

85 per cent. Analysis of the diagnostic failures by one or another of the roentgenologists revealed causes which can be divided roughly into three categories: (1) technically defective examinations, (2) malignant infiltrations camouflaged by the complex architectural components of a physiologically hyperactive breast in the younger adult, and (3) failure to correlate the clinical with the x-ray findings. The remaining failures were due to lack of experience in recognition of criteria of malignancy, especially the nature of calcareous deposits in malignancy.

TABLE I. ACCURACY OF X-RAY DIAGNOSES OF BREAST TUMORS (AMONG 210 CONSECUTIVE CASES, 47 CARCINOMAS AND 163 BENIGN LESIONS)

ROENTGENOLOGIST	FIRST INTERPRETATION WITHOUT CLINICAL FINDINGS				SECOND INTERPRETATION WITHOUT CLINICAL FINDINGS				THIRD INTERPRETATION WITH KNOWLEDGE OF CLINICAL FINDINGS			
	ERRORS BENIGN LESIONS		ERRORS MALIGNANT LESIONS		ERRORS BENIGN LESIONS		ERRORS MALIGNANT LESIONS		ERRORS BENIGN LESIONS		ERRORS MALIGNANT LESIONS	
	NO.	%	NO.	%	NO.	%	NO.	%	NO.	%	NO.	%
1	8	4.9	12	25.5	4	2.5	10	21.3	3	1.8	5	10.6
2	7	4.3	10	21.3	6	3.7	11	23.4	6	3.7	5	10.6
3	5	3.1	15	31.9	5	3.1	12	25.5	2	1.2	8	17.0

Illustrative case reports will help to indicate the pitfalls in the x-ray diagnosis of malignant breast lesions. There is no need to review here again the importance of technically perfect films in order to demonstrate the criteria of malignancy[4] (Fig. 1).

Malignant lesions may be overlooked in active dysplastic breasts.[5] This combination fortunately is infrequent. Women in this group complain of lumps which come and go quickly. Rarely, certain malignant processes, especially those that tend to have a lobular structure, can be confused easily with actively proliferating breast tissue. This is illustrated in the case of a 32-year-old woman who complained of slight pain in the right breast. Six months later, following a fall, a mass appeared which was observed for three months. Variations which occurred in its size were thought to be due to injections of testosterone. Finally, surgery was advised. The preoperative x-ray examination disclosed the presence of a smooth-walled mass, slightly smaller than the recorded clinical measurements. Such a discrepancy usually occurs with malignant lesions. The surrounding breast structures appeared complex, not unlike those due to active mazoplasia. No suspicion of malignancy was aroused by the x-ray appearance of this lesion, which was an adenocarcinoma among large lobular breast masses, some also carcinomatous and some merely hyperplastic (Fig. 2).

An example of the need for correlating the clinical with the x-ray findings is seen in the next case of a 65-year-old woman. She noted a mass 5 cm. in diameter for eight days in the upper outer quadrant of the left breast. Examination revealed dimpling of the skin with arm raising. The x-ray examination revealed a mass which was only irregularly defined and could not be completely differentiated from the surrounding breast tissue. It measured 1.5

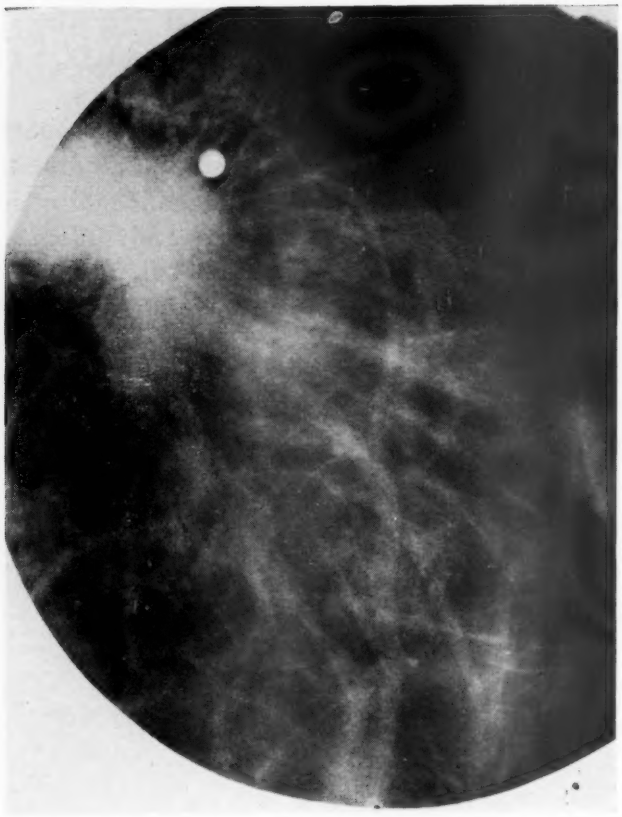
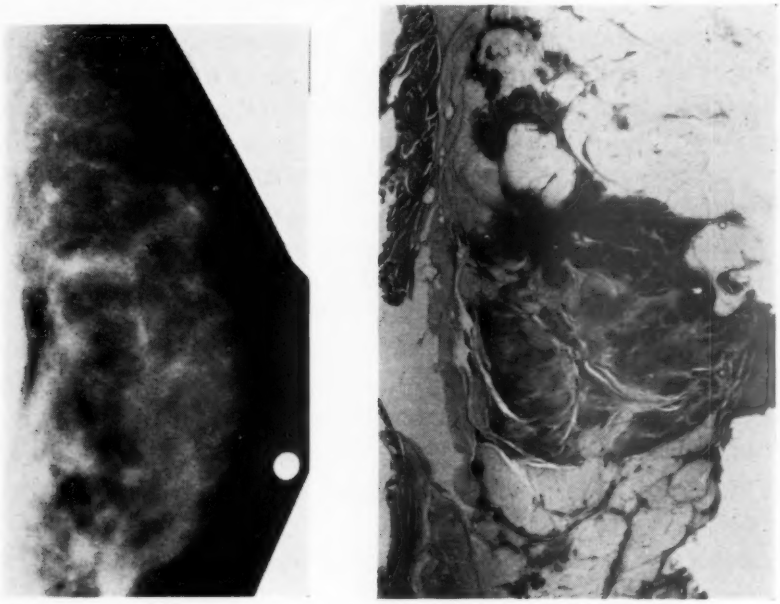


Fig. 1.—Carcinoma of breast. The x-ray criteria of malignancy require a perfect x-ray examination before they can be evaluated. The criteria in this case are the spiculated margins, the swollen perifocal trabeculae, the engorged veins, and the localized thickening of the overlying skin (seen with intense light transmission). A metal marker always is placed on the skin over the palpable mass to orient the roentgenologist.

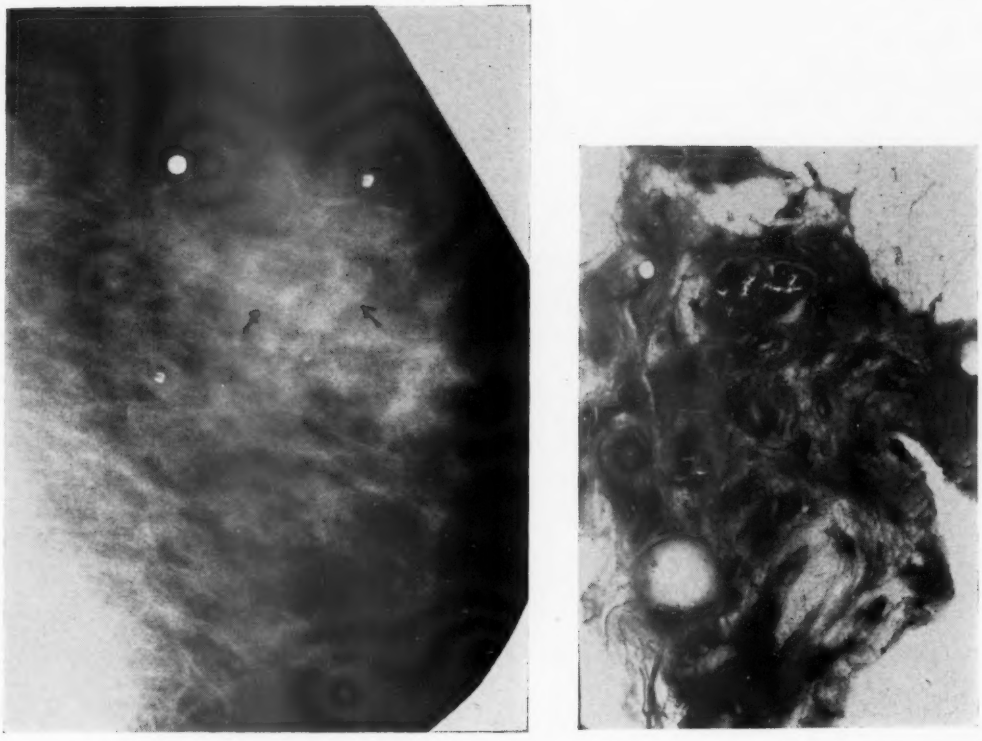


A. *B.*

Fig. 2.—Adenocarcinoma showing lobular spread.

A, The marker on the skin is over the palpable mass, the margins of which merge with the surrounding structures in a mazoplastic breast. The criteria of malignancy may be difficult or impossible to discern in such circumstances.

B, The tumor masses are continuous with the lobular structures, some of which are carcinomatous, some hyperplastic. All stages of transition from hyperplasia to carcinoma may be seen.

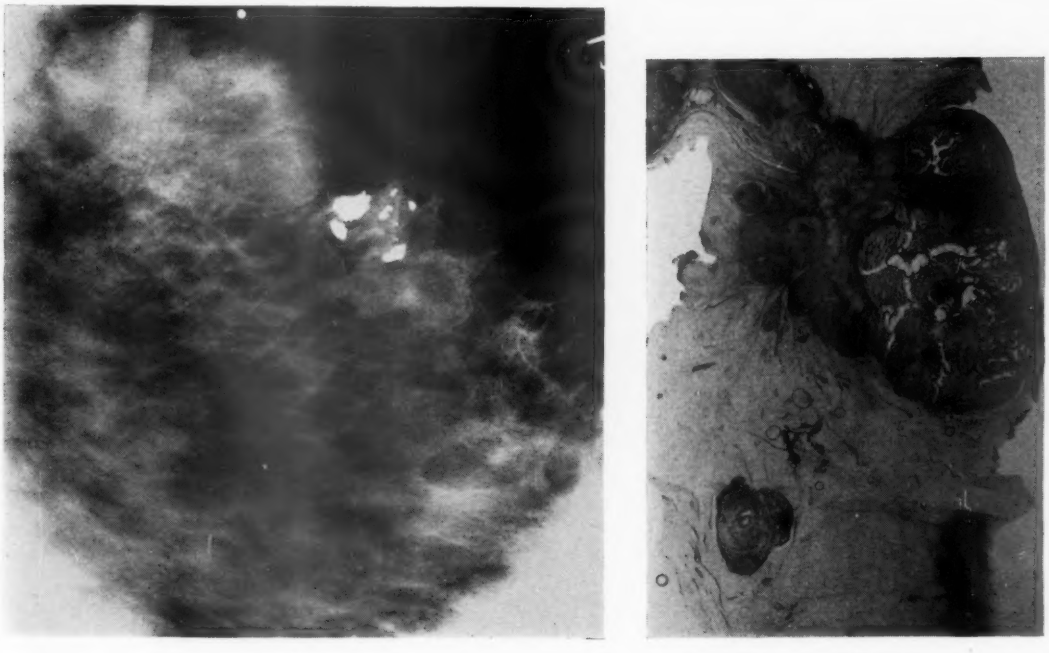


A. *B.*

Fig. 3.—Mucoid carcinoma spreading via dilated ducts.

A, The mass is poorly defined by the surrounding fibrotic tissue. Its diameter is 1.5 cm. less than that found by palpation, a common diagnostic discrepancy in malignancy.

B, Carcinoma showing mucoid and hyaline degeneration. It is surrounded by small cysts, some of which contain carcinomatous cells.



A. *B.*

Fig. 4.—Adenocarcinoma.

A, An oval mass (under skin marker) whose margins are smooth. The skin (as viewed under an intense light) is thick, and the perifocal trabeculae are swollen. Measurements of this mass are smaller than those made by palpation. Such are the x-ray findings to corroborate the clinical impression of a malignant lesion.

B, Adenocarcinoma with much fibrosis. A calcified fibroadenoma is near by, as seen in the x-ray film.

cm. less in diameter on the film than by palpation, an important criterion of malignancy. Otherwise, the x-ray appearance of this lesion resembled plasma cell mastitis.[6] This process was a mucoid carcinoma with hyaline degeneration. The surrounding mammary tissue was fibrotic and contained many cysts (Fig. 3).

Malignant lesions in fatty breasts of older patients may be so sharply defined as to simulate benign processes. In excellent films, the fine infiltrating extensions of such malignant tumors can be discerned by the roentgenologist only if he is particularly alert. Illustrating this situation is the case of a woman 60 years old, who had noted a lump in the left breast for four months. Clinical examination revealed the presence of an indurated mass 7 cm. in diameter. Peau d'orange skin covered a swollen breast. The x-ray examinations revealed a smooth-walled mass 5 cm. in diameter (less than the clinical measurements), resembling a cyst or fibroadenoma. Near by was another poorly defined mass containing some calcareous deposits suggesting a fibroadenoma or an old healed focus of fat necrosis. No changes were visible anywhere to suggest malignancy, yet the larger mass was a fibrotic adenocarcinoma quite well localized; the smaller near-by calcified tumor was a fibroadenoma (Fig. 4).

Calcification in malignant processes may be pathognomonic even when the x-ray appearances of the lesions otherwise may be misleading. Familiarity with this type of calcification, therefore, can be important.[7]

DISCUSSION

Experienced clinical pathologists agree that a reliable diagnosis can be made from the macroscopic appearance of most sections of the breast; otherwise, even the miscroscopic appearance often presents diagnostic difficulties. In a broad sense, these conclusions pertain to the x-ray appearance of most breast lesions, since they too resemble the macroscopic appearance of the freshly sectioned counterparts. Slices through a breast can furnish impressions such as can only be achieved by the roentgenologist with planigraphy. But planigraphy lacks the details necessary for accurate diagnosis of delicate breast structures. The roentgenologist must learn, therefore, to discern lesions in situ among their complex parenchymal environs. Sometimes this has advantages, since careful attention to the distorted secondary changes in the tissues around a breast lesion also has diagnostic value.

Most mistakes in x-ray diagnosis of malignant lesions occur in women under 45 years of age. After the menopause, lesions in the fatty breast are easily discernible, and the occasional false diagnostic interpretation arises because of the benign appearance of a localized, smoothly contoured malignant lesion. Attention to the clinical findings and to the secondary x-ray changes in the breast around such lesions then assumes much diagnostic importance.

Only one case of five in this series of cases had a malignant lesion. Biopsy was therefore carried out four times more often on breasts with benign than with malignant lesions. Some biopsies are either not carried out or they are postponed. In such cases, the x-ray examination furnishes a permanent record which can serve for comparison with serial re-examinations.

SUMMARY

A comparative review was made of the x-ray interpretations by three roentgenologists of 210 consecutive mammary lesions which were checked by the pathologist, either from biopsy or study of the whole breast sections. The x-ray diagnoses of three repeated readings were tabulated for accuracy among themselves by the same and among different radiologists and finally checked against the pathologic findings.

Of the 163 benign lesions, 97 per cent were correctly diagnosed, and of the 47 malignant lesions, 41 finally were accurately designated. Discrepancies among the x-ray diagnoses decreased with repeated interpretations, both among the repeated readings of the individual and between the readings of the several roentgenologists. Causes for errors in diagnosis of malignant lesions lurk occasionally in the complex architecture of the physiologically active breast in middle-aged women and less often among the isolated malignant, but benign-appearing, lesions of the fat postclimacteric breast. In the x-ray diagnosis of malignant lesions, careful attention must be given to technical details, diagnostic criteria, and the clinical findings. Examples of some pitfalls in diagnosis are given in this report.

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TRAUMA AND CAPILLARY RESISTANCE

A Clinical and Experimental Study

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IN THE constant endeavor to minimize the risks of surgical intervention and to render postoperative convalescence as short and undisturbed as possible, increased attention is being given to the pituitary-adrenocortical system as the principal factor in the stress phenomenon. Determination of urinary steroid excretion, of the level of the circulating eosinophils, and of the spontaneous or induced change in this level have been used as helpful adjuncts to clinical observation of the postoperative patient.

The purpose of this paper is to show that another method may be added to our armamentarium, namely, evaluation of the capillary resistance.

The resistance of the capillary wall as measured by the negative or positive pressure method and expressed in centimeters of mercury has been considered heretofore as a local phenomenon which may be influenced by many factors acting locally. This concept has changed entirely during the last three years. Clinical observations (Robson and Duthie[1, 2]) and animal experiments (Eichholtz and associates,[3] Kramár and Simay-Kramár[4]) have clearly demonstrated that capillary resistance is subject to hormonal influence. The principal hormone involved is cortisone. The correlation between capillary resistance and cortisone is so intimate that testing of the first may give information on the level of the latter. It has been shown that any decrease in the cortisone concentration in the organism (adrenalectomy, experimental depletion of the adrenal cortex in the late phases of stress response) is followed by a decrease in the capillary resistance, whereas any increase in the cortisone content of the body (administration of cortisone or ACTH, early phase of stress response) is accompanied by an increase in the capillary resistance.

It has been known since 1944 that trauma consistently causes the capillary resistance to increase after surgical interventions in the human being (Scarborough[5]), as well as following experimental crush in the rat, guinea pig, and mouse (Ungar[6]). In view of our recent knowledge of the endocrine background of the capillary resistance it seemed that a detailed study of these conditions would be highly desirable.

The present work consists of two parts. In the first, the effect of surgical intervention and of crush was studied on the capillary resistance of experimental

This work represents part of a project supported by the Cardiovascular Section of the Division of Research Grants of the United States Public Health Service and by the Creighton University School of Medicine.

Received for publication, Aug. 28, 1953.

animals. In the second part, the capillary resistance of human patients was observed during the postoperative course. The final aim was to correlate the results of clinical observations with the finding in the experimental animals.

METHODS

Capillary resistance was tested in both parts of this work by means of the negative pressure method. The device used consisted of a vacuum reservoir, a small transparent plastic suction cup (with an inner diameter of 7 mm.), and a mercury manometer with the necessary connecting tubing and stopcocks. Suction was supplied by an electric suction pump in the laboratory and by a two-way Potain hand aspirator in the wards. The abdominal skin was selected for the site of testing in the animals, the flexor aspect of the forearm in the human beings. The least negative pressure capable of eliciting capillary rupture and formation of petechiae was considered the value of the capillary resistance. Details of the technique, especially with regard to the precautions necessary for preventing erroneous readings or misinterpretation, have been described elsewhere (Kramár and Simay-Kramár,[4] Kramár, Peetz, and McCarthy[7]).

PROCEDURE

Part I.—Two types of experimental trauma were used, namely, surgical operations and crush of the hind limb.

1. Twenty albino rats (eleven males and nine females) of the Sprague-Dawley strain, weighing approximately 200 grams and maintained on Purina Dog Chow, were trained until they became fully accustomed to the restraint necessary for testing capillary resistance. Bilateral sham adrenalectomy was then performed under deep ether anesthesia. The lumbar approach was used and the adrenal gland was visualized but not touched. The operation was carried out in a deliberately circumstantial manner with the intention of producing an extensive and, at the same time, relatively standard tissue injury. For this purpose, muscle layers were cut through instead of being severed bluntly, length of incisions through skin and muscle, number of stitches, and duration of anesthesia and of the operation itself were standardized. A control base line of capillary resistance was determined prior to the operation, and the testing was continued at one-to-two-day intervals for about two months thereafter.

2. Five rats (two males and three females) were trained in the same manner, and then anesthetized with ether. One hind extremity was then crushed with a padded hammer. Testing of the capillary resistance was performed in the same manner as described previously.

3. Twelve rats (five males and seven females) were not subjected to the routine training procedure prior to the experiments. In addition, for testing capillary resistance they were tied to a board instead of being gently held by an assistant, as was done with the previous animals. In this way, a considerable emotional upset was created which gave rise to a typical capillary response causing the capillary resistance to drop after an initial rise and to remain at a

low level for about one week. Crush was applied during this latter period and the capillary resistance tested as described in the foregoing experiments.

Part II.—Ninety-four major surgical cases were selected at random, the criteria of selection being an anticipated hospital convalescent period of at least seven days. Forty-three men and fifty-one women, ranging in age from 17 to 78 years, were included. Eight cases were observed daily, and in eighty-six cases readings were obtained every other day. Initial readings in eighty-two cases were obtained on the day of surgery. In twelve cases, at least two daily preoperative readings were obtained. The type of anesthesia was considered relatively constant, as all patients received similar anesthesia consisting of Pentothal Sodium induction followed by ether, oxygen, and nitrous oxide mixture as required.

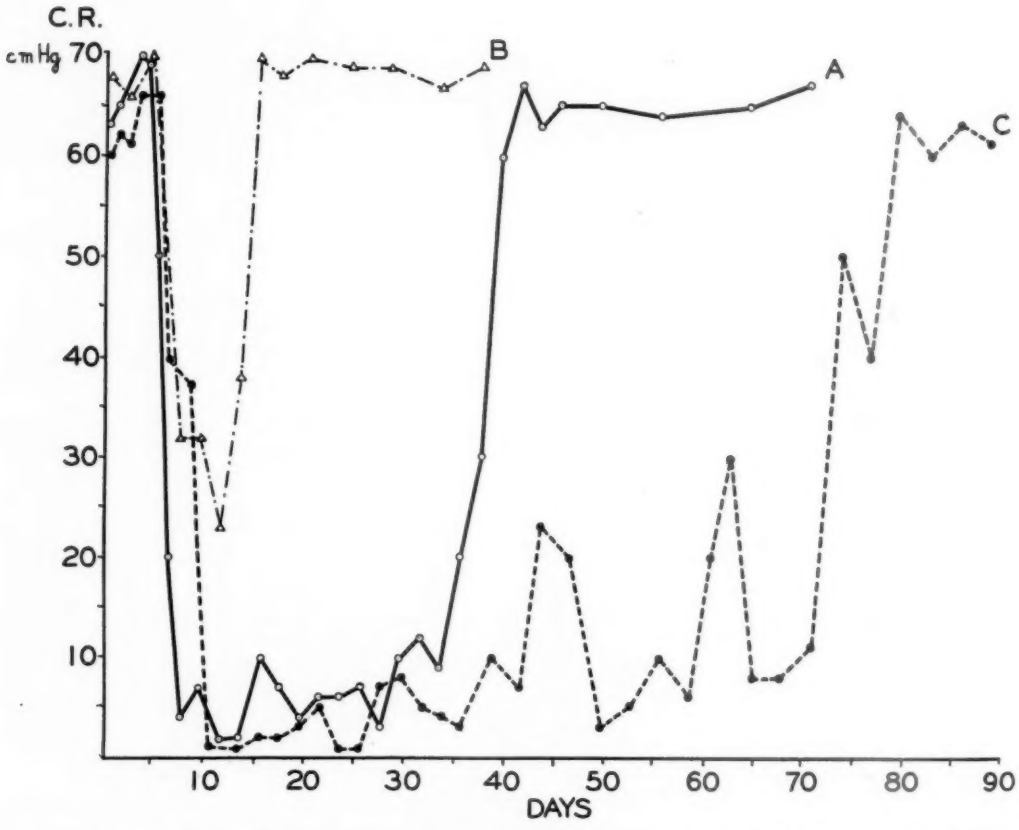

Fig. 1.—Patterns of capillary resistance change following surgical trauma (sham adrenalectomy) in the albino rat. *A*, Average pattern; *B*, accelerated or telescoped pattern; *C*, protracted pattern. The four phases of capillary response are best seen in the average pattern: initial rise, sudden drop, period of pathologically low resistance, and restoration to normal.

RESULTS

Part I (Animal Experiments).—

1. *Effect of sham adrenalectomy on previously trained rats:* As shown in Fig. 1, the changes occurring in the capillary resistance could be divided into four phases: (a) an initial rise immediately following operation, (b) a sudden

drop to pathologically low levels, (c) a period of abnormally low capillary resistance, and (d) restoration to normal. The length of the first phase was about the same in all the animals (one to two days) and the degree of rise was principally dependent on the preoperative level of the capillary resistance, as no marked increase could have occurred when the initial level was already high. Phases b, c, and d revealed more individual differences. On this basis, three patterns could be distinguished. That shown by the majority of the animals (fifteen of twenty) may be termed the *average pattern* (Curve *A* in Fig. 1).

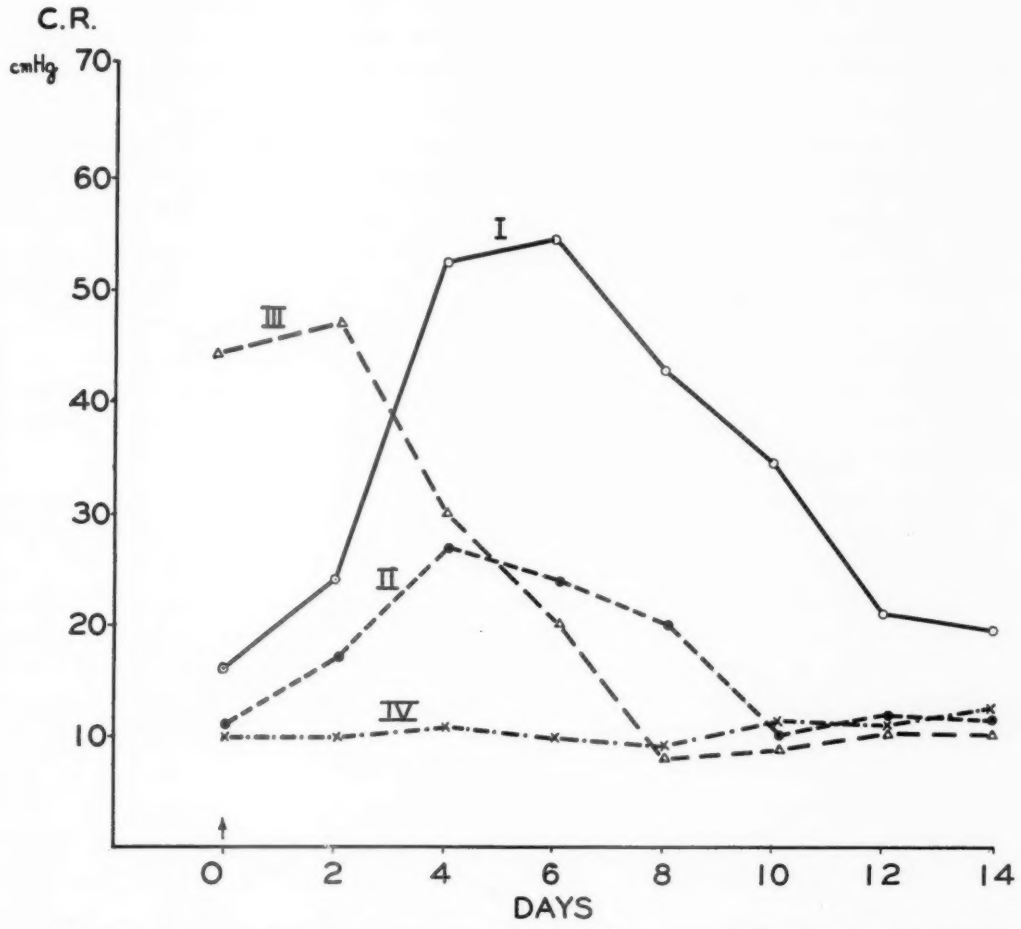

Fig. 2.—Patterns of capillary resistance change in the human being following surgery. *I*, Vigorous response (increase of capillary resistance by at least 20 cm. Hg); *II*, moderate increase (6 to 19 cm.); *III*, decrease of capillary resistance; *IV*, no change.

Here the drop of the capillary resistance was precipitous and the third phase, lasting about ten to fifteen days, was followed by rapid recuperation. The entire response had an average duration of thirty days. Three animals showed a capillary response that was shorter and less intensive. The drop in the second phase was moderate and the third phase very short. The entire duration of this *accelerated or telescoped pattern* was about ten days (Curve *B* in Fig. 1). Two rats disclosed another extreme in their capillary response: here the third phase

was of extremely long duration and the recuperation was interrupted by frequent relapses. The entire response lasted seventy to seventy-five days (*protracted pattern,* Curve *C* in Fig. 1).

2. *Effect of crush on previously trained rats:* The experiments in this category brought a uniform result. All five rats showed a change in the capillary resistance which was essentially similar to that seen after sham adrenalectomy. There was, however, some difference in the details. The first phase of the response was strikingly long (three to five days), during which time the capillary resistance was constantly at its highest level. The third phase, on the other hand, was much shorter, and recuperation took place very rapidly, hence, the entire response lasted, despite the longer first phase, only fifteen to twenty days.

3. *Effect of crush on untrained animals:* The low capillary resistance of these rats brought about by means of emotional stress prior to crush, rendered the first phase of the capillary response following crush very spectacular. The capillary resistance rose within the first twenty-four hours, the rise amounting to 20 to 60 cm. The duration of this high capillary resistance level, however, was very short and was followed by a typical drop in the second phase. In the course of the third and fourth phases, four rats disclosed an average pattern, whereas six rats had a protracted pattern. In one rat, the capillary resistance decreased only slightly after the marked initial rise, and thus the resulting pattern was rather ill defined. Similar "rudimentary" patterns were sometimes encountered following forced muscular exercise or cold stress (Kramár[8]).

Part II (Clinical Observation).—Although every case responded in an individual manner, all could be placed in one of four groups depending on the type and degree of change or changes found in the capillary resistance (Fig. 2). Group I included those cases in which an increase of at least 20 cm. Hg was noted in the capillary resistance during the observed period of the postoperative course. Group II was characterized by an increase of from 6 to 19 cm. Hg in capillary resistance. Group III included those cases in which the last recorded capillary resistance was lower than the initial reading and in which no pronounced increase of resistance was observed during the postoperative period. Group IV encompassed those cases in which there was no change in capillary resistance.

According to this classification, the distribution of our ninety-four cases was as follows:

Group	*Number of cases*	*Per cent*
I	33	(35)
II	31	(33)
III	15	(16)
IV	15	(16)

Having found that all the patients could be placed into four basic groups, an attempt was made to determine whether age, sex, and extent of the surgical procedure were causally related to the four groups.

The age of the patients was first correlated with the response, and it was found that no significant correlation could be made. However, when the sex

of the individual was correlated with type and degree of response, some interesting facts became evident (Table I). The majority of the male patients (51.3 per cent) were in Group I, the vigorous response category, whereas the largest percentage (45.3 per cent) of women fell into the less vigorously responding Group II. In contrast, Groups III and IV failed to reveal any sex difference.

TABLE I. CORRELATION OF TYPE OF CAPILLARY RESPONSE WITH SEX

GROUP	NUMBER OF PATIENTS	♂	♀
I	33	22	11
II	31	8	23
III	15	7	8
IV	15	6	9

Equally significant was the correlation of the degree of surgical intervention and type of capillary response (Table II). Arbitrarily, three degrees of surgical intervention were distinguished, depending on the amount of tissue disturbed at operation. Forty-nine cases of herniotomy, hysterectomy, and cholecystostomy were considered to have been subjected to the least traumatic type of intervention (''minor'' in Table II). Twenty-eight patients were subjected to gastric or intestinal resection, pulmonary lobectomy, suprapubic prostatectomy, gastroenterostomy, and amputation, that is, interventions assumed to be connected with a moderately severe degree of tissue damage (''moderate'' in Table II). Seventeen patients were subjected to the most severe and extensive surgical procedures, such as combined abdominoperineal resection, radical mastectomy, arthroplasty, spinal fusion, and any other type of procedure followed by unusual complications (''marked'' in Table II).

TABLE II. CORRELATION OF TYPE OF CAPILLARY RESPONSE WITH DEGREE OF SURGICAL INTERVENTION

DEGREE OF TISSUE TRAUMA	NUMBER OF PATIENTS	TYPE OF CAPILLARY RESPONSE			
		GROUP I	GROUP II	GROUP III	GROUP IV
Minor	49	10	22	10	7
		20.4%	44.9%	20.4%	14.3%
Moderate	28	14	4	5	5
		50.0%	14.4%	17.8%	17.8%
Marked	17	9	5	—	3
		52.9%	29.4%		17.7%

It is seen that whereas the dominant type of response among our patients subjected to minor degree of tissue trauma was that of Group II, the majority of patients inflicted with a moderate or marked degree of tissue damage showed the Group I type of response. It is interesting to note that 14 to 18 per cent of patients exhibit no definite increase in the capillary resistance regardless of the type of surgery performed.

DISCUSSION

The change in the capillary resistance found in the experimental part of this work following two kinds of tissue injury, surgical operation and crush, was similar to that encountered in previous works after cold stress, forced muscular

exercise (Kramár[8]), and after severe emotional upset (Kramár, Peetz, and McCarthy[7]). This uniformity of response suggests that we are dealing with a nonspecific phenomenon that may have a common, nonspecific underlying mechanism. It is believed that this capillary response with its four well-defined phases is part of the general stress syndrome as described by Selye.[9] In view of the previous work discussed in the introduction of this paper concerning the hormonal regulation of the capillary resistance, it seems that the initial phase of this response, the rise in the capillary resistance, is the result of ACTH discharge and consecutive adrenocortical activation, whereas the second and third phases, termed "capillary crisis," are due to a hypofunction of the adrenal cortex, presumably caused by its nonresponsiveness (depletion?). The fourth phase, then, may represent one of the many manifestations of the restoration process taking place in the organism.

The clinical part of this work demonstrates that the change in the capillary resistance following surgery is much more complicated than as described by Scarborough.[5] On the basis of two readings, one three hours preoperatively, the other twenty-four hours postoperatively, this author stated that all the twenty-three cases observed evidenced a definite increase in the capillary resistance. He further maintained that this increase appeared to be independent of the age and sex of the patient as well as of the nature of the operation performed.

While observing a greater number of patients during a longer period of time, it was found, first, that this capillary response is subject to great individual variations. In the final analysis, four patterns could be established that would encompass all these variations. Second, a definite sex difference was found in that men are prone to develop more vigorous response to the same degree of tissue damage. Third, a definite relationship was found between type of response and kind of surgical intervention. In general, as the amount of tissue involved increases, the more marked becomes the increase in the capillary resistance.

Before any attempt is made to evaluate the meaning and significance of these findings, it seems necessary to correlate them with the results of the experimental study. The comparison shows a common feature and a discrepancy.

There is a tendency, in both human being and experimental animal, to develop certain patterns in the capillary response. There is no doubt that capillary response may be influenced by many factors. In experimental animals subjected to other types of stress (cold, forced muscular work), these factors have been fairly well established (Kramár[8]). We know that it is not so much the type of stress as its intensity that is decisive and, furthermore, that previous diet, repetition of stress (training), and individual differences are also modifying factors. The effect of some of these factors was seen in the present work. The role of individual differences was well demonstrated in the experimental part in which rats were subjected to sham adrenalectomy. Despite carefully standardized experimental conditions, these animals displayed three patterns of response. The individual differences were also seen in the clinical material. The patients, although subjected to the same kind of operations, exhibited four

patterns of response. On the other hand, the influence of the intensity of stress, that is, of the type of operation involving various degrees of tissue damage, upon the capillary response was also demonstrable: the greater the stress, the higher the number of patients with Group I (very pronounced) response.

The discrepancy between clinical and experimental observations lies in the fact that whereas the majority of experimental animals disclosed a typical change in the capillary resistance with four well-defined phases, this "full-blown" capillary response was only exceptionally encountered in the human being. As a rule, the postoperative patient showed only fragments of the change displayed by the experimental animal, although these fragments are well recognizable. The Group I and II type of response represents the first phase of the capillary response in the rat; Group III corresponds to the second and third phases. No match was encountered in the present animal experiments for the nonresponsive Group IV of the patients. Nevertheless, as this refractory type was repeatedly found in other stress experiments it may also be expected following trauma provided that a greater number of animals is used than in the present work.

It is believed that this difference between clinical and experimental observations has a phylogenetic basis. The albino rat, the animal used in these experiments, has a sheltered life and is hardly exposed to any serious stress. Consequently, its pituitary adrenocortical axis is very sensitive and its response to stress prompt and alarming, in sharp contrast to the well-trained system and, therefore, more sluggish response in the human being.

In reviewing the recent surgical literature, one gathers the impression that a prompt pituitary-adrenocortical response has been regarded as obligatory for a satisfactory and uneventful postoperative recovery. In our series of ninety-four cases we fail to recognize this direct relationship. Apparently, some 14 to 18 per cent of individuals do not exhibit increased pituitary-adrenocortical activity as measured by capillary resistance but, nevertheless, make a very uneventful recovery. On the other hand, excessively high capillary resistance values may be found in patients whose postoperative course is far from satisfactory. Two cases serve as examples.

A 45-year-old woman was subjected to gastric resection. No elevation in the capillary resistance occurred through the convalescence. On the tenth postoperative day a standard Thorn test was performed with entirely negative results. The postoperative course of this patient was completely uneventful.

A 78-year-old man, following a combined abdominoperineal resection, exhibited a marked increase ($>$ 70 cm. Hg) in capillary resistance. For six weeks the capillary resistance remained high, and the postoperative course was very complicated. The wound disrupted on the fifth postoperative day; repeated episodes of partial bowel obstruction requiring Levin suction further clouded the issue, and a pre-existing asthma continued to flare. On the thirty-fourth postoperative day a marked fall in the capillary resistance (from 70 to 28 cm. Hg) occurred; two days later a generalized edema appeared and the patient expired rapidly.

We are far from implying that no correlation exists between postoperative recovery and pituitary-adrenocortical function. We wish to emphasize solely

that such a correlation is more complex than is generally appreciated. There are several reasons for the difficulty encountered in the evaluation of this correlation.

The first concerns the pituitary-adrenocortical function per se, irrespective of the method of testing. We really do not know what is the most favorable type of postoperative pituitary-adrenocortical response. Our still limited knowledge of the basic mechanism of wound healing and the capacity of the organism to meet stress may fully account for this shortcoming.

In addition, there are other reasons originating in the particular method used in this work for testing pituitary-adrenocortical function. We do not know yet whether cortisone is the sole hormone regulating capillary resistance or, as on the basis of some current work it may be suspected (Kramár[10]), whether the level of the capillary resistance reflects the balance of several hormonal functions. We also begin to realize that both low and high capillary resistance may have more than one interpretation. Assuming that cortisone alone is responsible for the actual level of the capillary resistance, a low resistance may, for instance, indicate that adrenocortical function is at its leisure because there is no need for increased activity, but also that the adrenal cortex is incapable of increased activity. By the same token, a high level in the capillary resistance may herald a vigorous adrenocortical function resulting from stimuli favorable for the organism (for example, physical activity after long sedentary occupation, effect of sunshine after a long, gloomy winter, recreation), but also a desperate struggle against a stress of abnormal intensity and duration. A sudden drop of the resistance in this latter condition may mean that the stress ceased to exist but also that the organism, owing to adrenocortical exhaustion, is unable to cope with it.

SUMMARY

The effect of trauma on the capillary resistance was studied in animal experiments and on surgical patients in the postoperative convalescence.

Groups of albino rats were subjected to surgical operation, others to crush. A uniform response was found consisting of four phases: initial rise of the capillary resistance, sudden drop, a period of abnormally low level, and restoration to normal. In view of the similarity of this response to that encountered after various other types of stress, the described change in the capillary resistance is considered a part of the nonspecific stress syndrome. An attempt has been made to evaluate these changes with reference to our recent knowledge of the endocrine regulation of the capillary resistance.

Patients during the postoperative convalescence showed the full-blown rat-pattern of the capillary resistance change only exceptionally. Most of them exhibit only fragments, that is, one or the other phases of the response typical of the rat. A phylogenetic explanation is given for this difference.

The capillary response in the human being following surgery is related to the amount of tissue involved in the procedure, sex of the patient, and individual characteristics.

The question of correlation of the capillary response to the clinical picture of the recovery phase is still open to discussion and requires further investigations.

Acknowledgment.—We are indebted to Dr. C. M. Wilhelmj of the Creighton University School of Medicine for his constant interest and valuable assistance.

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BLOOD CULTURE STUDIES IN NORMAL DOGS AND IN DOGS IN HEMORRHAGIC SHOCK

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INTEREST in the interrelationships between shock and infection has been reactivated with the recent reports of a rapid shocklike state produced by bacteremia in animals[1, 2] and in human beings,[3-5] the observation that the mortality rate from a standard hemorrhagic shock procedure is reduced in dogs by oral pretreatment with aureomycin or neomycin,[6] and the finding that, in shock, there is an increased blood level of a specific bacterial toxin initially placed in the intact intestine of a dog.[7] Such data suggest that bacteria may play a role in profound shock, even within the breadth of time ordinarily considered too brief for bacterial complications.

In the present study, blood cultures were taken from dogs during the course of experiments on hemorrhagic shock. This communication reports the results obtained from initial and terminal blood cultures in dogs in shock and in control dogs not in shock.

METHODS

Animal Preparation.—This experiment utilized seventy-three mongrel dogs, not selected for sex, and weighing approximately ten kilograms. Twenty-four dogs served as controls, and forty-nine were subjected to a state of hemorrhagic hypotension. Prior to experimentation, the dogs were held for thirty days for standardization of environment, deworming, and immunization against canine distemper. Experiments were then performed in an air-conditioned operating room. Each dog was lightly anesthetized with ether. The skin of the groin was shaved and prepared with surgical detergent and alcohol, and the femoral vessels were surgically exposed, employing strict aseptic precautions. Heparin (6 mg. per kilogram) was injected intravenously, and all initial blood cultures were taken from the exposed femoral artery. In the control dog, the femoral artery was ligated, and in the dog to be bled, a 13 gauge cannula was inserted into the right femoral artery and connected to a sterile reservoir system, as described elsewhere.[7] The animals were then allowed to recover from anesthesia, and no further ether was employed. The control dogs were returned to the cage for later sampling, and the "shock" dog was bled until the pressure in the system was 30 to 40 mm. Hg. The mean volume of blood withdrawn to produce this state of hypotension was 52 ml. per kilogram, which figure is consistent with

Received for publication, Sept. 29, 1953.

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that reported by other investigators in this type of preparation.[8] Duration of the period of hypotension ranged from one hour to six hours, with the period in twenty-four of the forty-nine dogs extending beyond five hours. Final blood culture was taken in the shock dogs from the femoral artery, just prior to death in most instances. Final samples were withdrawn through the surgically prepared skin from the cubital or jugular vein of the control dogs at a corresponding time.

Bacteriologic Methods.—Two cultures were obtained from each dog, an initial and a final specimen. Therefore, ninety-seven cultures were from dogs not in shock (including forty-nine initial specimens from the shock group) and forty-nine cultures were from dogs in hemorrhagic shock. Ten milliliters of blood was injected into a sterile rubber-stoppered vaccine bottle containing 50 ml. of trypticase soy broth with 0.1 per cent sodium citrate and 0.1 per cent agar. Samples for staining and isolation were removed after one, two, seven, and fourteen days of incubation at 35° C. These samples were inoculated into thioglycollate medium for the cultivation of microaerophilic and anaerobic organisms, and streaked on blood agar and trypticase soy agar plates for the cultivation of aerobic organisms. Specimens of bacterial growth in thioglycollate broth were streaked on blood agar plates which were incubated in anaerobe jars for forty-eight hours. Identification was based on standard criteria.*

RESULTS

The incidence of positive blood cultures for the initial and terminal samples for the two groups is summarized in Table I.

TABLE I. INCIDENCE OF POSITIVE BLOOD CULTURES

GROUP	INITIAL	FINAL
Control dogs (24)	4 (16.7%)	8 (33.3%)
Shock dogs (49)	9 (18.4%)	11 (22.3%)

Inasmuch as the initial samples from the shock group were drawn from dogs still in a control state, these cultures can be grouped with the cultures from the dogs in the control group in order to determine the incidence of sterile versus positive cultures in control and shock states. This analysis is presented in Table II.

TABLE II. INCIDENCE OF STERILE AND POSITIVE BLOOD CULTURES

GROUP	STERILE	POSITIVE
Dogs not in shock (97 cultures)	76 (78.3%)	21 (21.6%)
Dogs in shock (49 cultures)	38 (77.6%)	11 (22.3%)

**Escherichia coli* and *Paracolobactrum aerogenoides* were identified by Gram stain and characteristic biochemical reactions.

Corynebacterium species were identified by the characteristic appearance of the colonies on tellurite serum agar.

Actinomyces species were identified by microscopic appearance and morphology in thioglycollate broth and on blood agar plates.

Micrococci and streptococci were identified by colony morphology and Gram stain. No attempt was made to type according to Lancefield classification.

Clostridium perfringens was identified by Gram stain and double zone of hemolysis on blood agar.

Table II demonstrates an essentially identical incidence of sterile and positive blood cultures between samples drawn from dogs in shock and those in a control state.

Only in four (16.7 per cent) of the twenty-four control dogs and in five (10 per cent) of the forty-nine shock dogs was the final culture positive when the initial culture was negative, indicating no greater predilection for subsequent positive blood cultures in the state of shock.

Analysis of bacteria isolated from the positive blood cultures is presented in Table III.

TABLE III. BACTERIA ISOLATED FROM BLOOD CULTURES

	CONTROL CULTURES		SHOCK CULTURES	
	NUMBER	PER CENT OF 21 POSITIVE CULTURES*	NUMBER	PER CENT OF 11 POSITIVE CULTURES†
Clostridium perfringens	7	33	5	45
Corynebacterium species	5	24	2	18
Micrococcus pyogenes var. *albus*	1	5	2	18
Hemolytic streptococcus	1	5	0	0
Nonhemolytic streptococcus	3	14	1	9
Escherichia coli	2	10	2	18
Paracolobactrum aerogenoides	3	14	1	9
Bacillus subtilis	1	5	0	0
Actinomyces species	1	5	0	0

*Total exceeds 100 per cent because two species of organisms were isolated from three cultures.

†Total exceeds 100 per cent because two species of organisms were isolated from two cultures.

In a small number of cultures of mesenteric lymph nodes, intestinal organisms were found which were not present in the peripheral blood of the same animal.

DISCUSSION

The bacteria cultured, as shown in Table III, were predominantly *Clostridium perfringens* and Corynebacterium species (diphtheroids). The flora isolated from the two groups were similar.

The finding of a similar incidence of positive blood cultures in shock and control states, of course, does not necessarily indicate that bacteria may not play a role in potentiating the pathophysiology of shock. The gross technique of blood culture yields only an index as to the presence of living bacterial cells, and not to the toxins or products of bacteria, which are the agents responsible for the deleterious consequences of infection. In this regard, we previously found that the concentration of a specific bacterial toxin, initially placed in the intact intestine, was higher in the peripheral blood of dogs in shock than in control dogs.[7] At least it would appear from these data that the incidence of bacteremia in shock is no greater than in normal dogs, so that further investigation into the role of bacteria in shock will require more specialized techniques capable of measuring toxins.

The application of these data to the problem of shock in human beings is precluded because the bacteriology of the dog is vastly different from that of the human being. While bacteria can be cultured from the liver of dogs in

35 per cent of biopsies, for example, less than 2 per cent of human liver biopsies are positive.[9]

SUMMARY

Of ninety-seven bacterial cultures taken from the peripheral circulation of control dogs 21.6 per cent were positive. Similarly, 22.3 per cent of forty-nine cultures taken from dogs in terminal hemorrhagic shock were positive. The predominant organisms cultured from both groups were *Clostridium perfringens* and Corynebacterium species (diphtheroids). Interpretation of these data is limited by the difference in the bacteriology of the dog from that of other mammalian species, and by the inadequacy of blood culture to provide a means by which the harmful effects of bacteria may be measured.

Acknowledgment.—We are indebted to Lieutenant Colonel E. J. Pulaski, Lieutenant Derwin Ashcraft, and Mrs. Jane S. Poole for their valuable aid in this study.

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GALL BLADDER TRACTION

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A MARKEDLY thickened, edematous gall bladder, or one which is filled solid with stones, is rather difficult to apply traction to with the usual clamps. The clamp will invariably cut through, resulting in a spillage of considerable bile or pus, or the clamp will slip off.

As traction is extremely important in attaining adequate exposure of the cystic common duct area, some other means had to be found.

Application of figure-of-eight sutures at different levels in the gall bladder seemed quite satisfactory. This has been carried out in a fairly large series of cases during the past two years and has proved to be even superior to clamps.

Fig. 1.

Fig. 2.

The amount of spillage due to the needle holes is minimal and rarely of any serious danger. The photograph and picture demonstrate the technique.

The No. 0 chromic suture is preferred over silk or cotton, as it is less likely to cut through the wall of the gall bladder. The lower suture offers an excellent means of pulling a dilated Hartmann's pouch away from the common duct, thus lessening the danger of injury to the common duct.

Received for publication, Sept. 22, 1953.

Case Reports

SPLENIC HEMANGIOMA WITH SUBSEQUENT FATAL HEMANGIOSARCOMA

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HEMANGIOMAS are probably the most common primary benign tumors of the spleen, but their incidence cannot be determined accurately because of the variable terminology used in reporting cases. Vascular splenic lesions have been grouped together as hamartomas, endotheliomas, or angiomatous cysts; however, none of these classifications is entirely satisfactory, for investigators have collected case reports according to their individual interpretation, and cases are so rare that few have reported more than a single instance.[1-6]

Splenic hemangiomas are extremely uncommon, and most of them are found at autopsy. Schottenfeld and Wolfson[7] found four angiomas of the spleen in 2,800 autopsies, and Thiele[8] discovered three splenic angiomas in 19,000 autopsies. In 1942, Pines and Rabinovitch[9] were able to collect only forty-two cases, and 52 per cent of these were discovered incidentally at autopsy. Most of the previously reported vascular lesions of the spleen were benign histologically, and the majority were single and small.[10-12] Doubt has been expressed that vascular lesions in the spleen and elsewhere should be referred to as tumors since it cannot be proved that they are not congenital vascular abnormalities which became clinically significant because of infectious, hormonal, or mechanical factors. Splenic angiomas are of no clinical importance unless they are malignant or of sufficient size to enlarge the organ. The size of the spleen, the multiplicity of the lesions, and the histologic appearance of the tissue warrant the use of the term tumor in the case to be described.

CASE REPORT

A 23-year-old Negro woman was admitted to Garfield Memorial Hospital on Nov. 15, 1949, having been referred from the Obstetrical Clinic for investigation of a mass in the upper left quadrant of the abdomen and a nodule in the right supraclavicular fossa. She was five and one-half months pregnant.

Roentgenograms revealed clear lung fields, enlarged mediastinal lymph nodes, and an enlarged spleen which extended nearly to the iliac crest. Bone marrow and numerous clinical laboratory examinations were reported to be within normal limits. Needle biopsy of the spleen yielded only fibrous tissue, blood, and a small amount of normal lymphoid tissue.

Removal of the right supraclavicular mass was attempted, but it was found to be a thin-walled, fluid-containing cystic structure which was evacuated and only partially removed. This proved to be a cystic hygroma which, as seen on microscopic examination, was lined by a thin, hyalinized, fibrous membrane. Outside of this was an incomplete layer of fibrofatty tissue supporting small aggregates of lymphocytes and lymphatic channels lined by flattened endothelial cells.

Received for publication, July 27, 1953.

Because of the roentgenographic findings in the mediastinum and the enlarged spleen, a presumptive diagnosis of lymphoma was made, and a histologic diagnosis could not be established except by thoracotomy. A test dose of irradiation was administered, and the patient received a calculated tumor dose of 1,100 r over the mediastinum between Dec. 27, 1949, and Jan. 10, 1950. Roentgenograms taken on Jan. 31, 1950, showed no essential change in the size of the mediastinal masses.

On March 2, 1950, the patient gave birth to a normal, living male infant after eight hours of labor. No difficulty was encountered in the delivery, and she was discharged on the fourth post-partum day.

Three months later, May 2, 1950, the patient was readmitted and the spleen was removed through a left rectus incision. Exploration of the abdominal cavity at the time of operation revealed no adhesions about the spleen, no accessory splenic tissue, and no other gross abnormalities. The patient made an uneventful recovery and was discharged from the hospital on the eighth postoperative day.

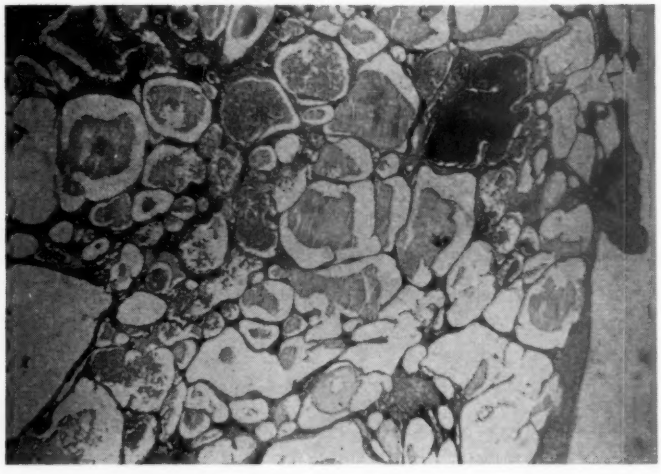

Fig. 1.—Photomicrograph of a typical area of hemangioma of the spleen. (Hematoxylin and eosin. ×75; reduced ½.)

The spleen weighed 3,065 grams and measured 36 by 23 by 12 cm. The surface was nodular because of numerous soft subcapsular dark red masses measuring from 0.5 cm. to 3 cm. in diameter. Cut section of the spleen showed a honeycombed structure composed of blood-filled cysts separated by thin, dark red, fibrous strands. The cysts varied in size from 0.1 cm. to 5 cm. and contained either blood or amber fluid. No recognizable splenic tissue was present in any part of the organ.

Microscopic sections of blocks removed from numerous parts of the spleen showed a similar structure. The tissue was composed almost entirely of blood-filled spaces separated by wide bands of hyalinized, fibrous tissue and supporting strands of lymphoid cells devoid of germinal centers. The blood-filled spaces varied greatly in size, and the larger ones were multiloculated. All the spaces were lined by normal, flattened endothelial cells.

The patient was readmitted Feb. 12, 1952, complaining of hemoptysis and substernal pain of four months' duration. Physical examination was essentially negative except for dullness and moist râles over the entire left lung. Bronchoscopy showed that the blood was coming from the left lower lobe, and bronchial washings were negative for acid-fast organisms and tumor cells. Roentgenograms showed the left hilar mass to be essentially unchanged and the right paratracheal lymph nodes to have increased in size slightly. Bronchograms demonstrated a slight narrowing of the left main bronchus due to pressure from the mediastinal mass. No areas of ulceration were demonstrated to account for the pulmonary hemorrhage.

Laboratory procedures were helpful only in revealing anemia, which was combated by repeated transfusions.

On Feb. 28, 1952, a left pneumonectomy was performed,* and two groups of mediastinal nodes were removed. The lung weighed 520 grams. Its surface was smooth except for numerous soft, dark red, bullous nodules averaging 1 cm. in diameter. On cut section the bronchi were blood-filled and the normal spongy lung tissue was almost completely replaced by irregularly shaped, blood-filled cysts, some of which communicated with the small bronchi. The mediastinal lymph nodes, which averaged 3 cm. in greatest dimension, collapsed when sectioned, and were composed of large, thin-walled cysts containing blood or yellow fluid.

Most of the blood-filled cysts in the lungs were microscopically similar to those in the previously removed spleen, and were lined with normal endothelium.

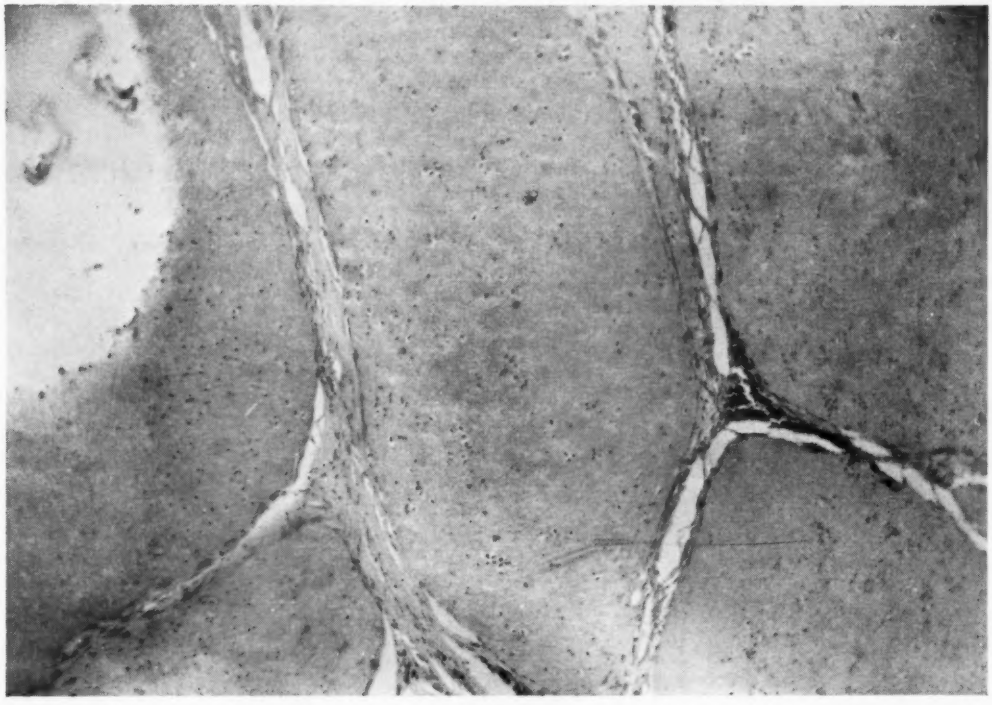

Fig. 2.—Photomicrograph of a mediastinal lymph node showing an area of hemangioma. (Hematoxylin and eosin. ×175; reduced ¼.)

Much of the lung parenchyma separating the vascular cysts was replaced by hyalinized fibrous tissue. In addition to the cavernous hemangiomas, numerous cystic structures, composed of a distinctly different type of tissue, were scattered throughout the lung. These cysts were small, irregular, and lined by large, cuboidal hyperchromatic cells. There was a suggestive papillary structure in some areas, and numerous rounded groups of enlarged endothelial cells projected into the lumina of the small, endothelial-lined spaces. Mitoses were present but extremely rare. The adjacent fibrous stroma showed widespread invasion by malignant cells, forming poorly differentiated blood vessels devoid of blood.

The resected mediastinal lymph nodes were composed almost entirely of cysts similar to those in the lung. Some of the cysts were surrounded by thick bands of fibrous tissue and were lined by normal, flattened epithelium, while in other adjacent cysts the lining cells were obviously malignant.

Postoperative roentgenograms showed numerous ill-defined masses in the right lung and slight enlargement of the mediastinal nodes on the right. Following the operation, the

*By Dr. R. G. Klepser.

patient continued to have hemoptysis, and expired on the third postoperative day. At autopsy the right lung weighed 910 grams. Its appearance was similar to that of the excised left lung. Bullouslike, dark red, soft, subpleural nodules and the blood-filled cysts occupied approximately one-half the lung parenchyma. There were many enlarged, ovoid mediastinal and periaortic lymph nodes composed mainly of bloody cysts. The liver weighed 1,550 grams and its surface was scattered with subcapsular multilocular cysts containing blood. Similar structures, present throughout the entire liver, averaged 1 cm. in diameter. In both ovaries there were small, soft, dark red, compressible areas measuring up to 1 cm. Cut section showed these to be blood-filled cysts. There was no involvement of the brain, bones, or any of the remaining viscera by the angiomatous process.

Microscopic sections of the right lung, liver, ovaries, and mediastinal and periaortic lymph nodes showed both benign and malignant vascular tumors similar to those described in the excised left lung and mediastinal nodes.

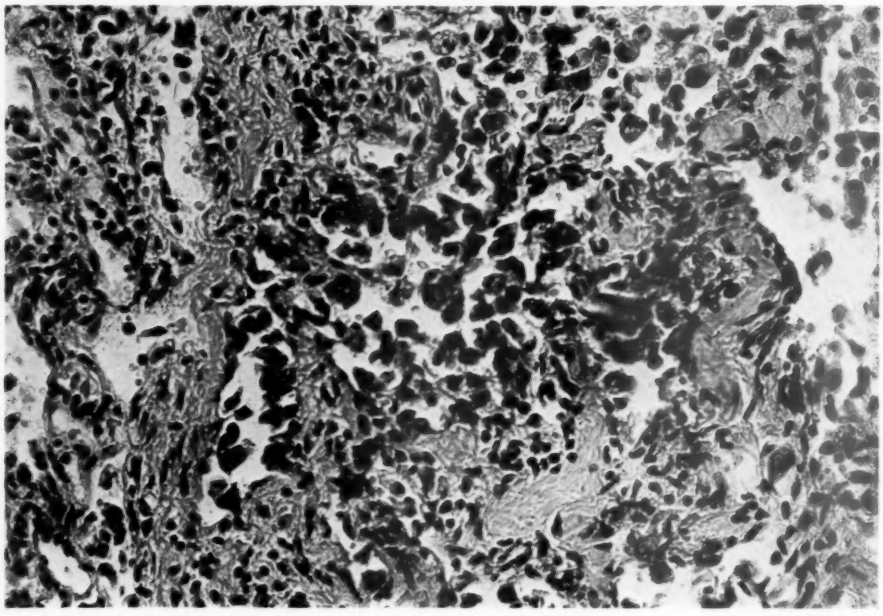

Fig. 3.—Photomicrograph of the same lymph node as in Fig. 2 showing angiosarcoma. (Hematoxylin and eosin. ×175; reduced ⅓.)

DISCUSSION

It is difficult to evaluate all aspects of the disease in this case and to classify it properly. Most of the vascular lesions of the spleen are benign cavernous hemangiomas, but occasionally, as in the case being reported, they are more complex lesions and are associated with benign or malignant vascular lesions elsewhere. Our case demonstrates some features similar to those reported in earlier cases. In one other reported case, metastases were found in lymph nodes,[13] and metastatic tumor was present in the ovaries in one instance.[14] One patient was pregnant at the time of the discovery of the splenic tumor.[15] The size of the splenic tumors in the comparable cases reported has varied from a few centimeters to a tumor occupying the entire organ and weighing as much as 5,800 grams.[16]

This condition is not always fatal, for several patients with angiosarcoma of the spleen were reported to be living after splenectomy, but very few have been followed for an adequate period.

Numerous explanations of the disease process in this case are possible, but none is entirely satisfactory. The primary site of the angiosarcoma is not known.

The case is reported as a hemangioma of the spleen despite the fact that only sixteen sections, taken at random in a very large organ, were examined histologically. It is recognized that malignant portions might have been present in areas not examined microscopically. A thorough gross examination did not reveal tissue different from that selected for microscopic examination. It could be postulated that the primary site of the tumor was in the spleen and was not included in the numerous sections taken for microscopic examination. This viewpoint is attractive, for the widespread angiosarcoma could be considered as metastatic tumor. However, this does not explain the presence of benign nemangiomas in some organs involved by angiosarcoma and the fact that the liver was grossly uninvolved at the time of splenectomy. Furthermore, there is roentgenographic evidence that mediastinal lymph nodes were involved two years before death, and the thick, hyalinized walls of the blood-filled spaces in all of the involved organs indicate that the benign hemangiomas had been present for a long time.

There is insufficient evidence to consider the liver as the primary site of the angiosarcoma because of the small, multiple foci of tumor in this organ and also because it would be difficult to explain the presence of metastatic tumor in the abdominal lymph nodes. Primary angiosarcoma of multiple lymph nodes may be a possibility, but does not appear probable in this case, and no report of such a case was found in the literature.

The lungs are dismissed as the primary site of the tumor for they were the organs least affected by the malignant process, and were clinically the last organs to become involved.

Probably the most logical diagnosis is multiple visceral cavernous hemangiomas with simultaneous malignant transformation of certain portions of the lesions in numerous organs.

There is no satisfactory explanation for all of the features of the disease in this case, for none can be proved, and most of the previously reported cases have a similarly questionable primary site. The confusing aspects of this bizarre disease cannot be clarified until a larger number of similar cases are reported or until more is known about the etiology of cavernous hemangiomas and angiosarcomas.

SUMMARY

A case of fatal angiosarcoma is reported in which the malignant disease became apparent two years after the removal of a large splenic hemangioma.

The literature relative to hemangiomas of the spleen is reviewed and discussed briefly.

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ABDOMINAL PREGNANCY WITH FETAL AND MATERNAL SURVIVAL

Report of a Case

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THE survival of the mother and a normal child following abdominal pregnancy is sufficiently rare to justify the report of another case.

Broomes[1] reviewed 343 cases of abdominal pregnancies, including his own case, and reported that there were only 85 cases in which both mother and child survived, and of these, only 33 children showed no demonstrable evidence of neurological or mental disturbances.

Jarrett[2] stated that 565 cases of abdominal pregnancies were recorded between 1809 and 1946. He added two cases, with the survival of both mothers and one child.

Review of recent literature[3-15] showed 24 additional cases with survival of 22 mothers and 13 infants, with 12 of the latter normal in all respects.

The management of the placenta varied. It was completely removed, or it was marsupialized and cavity-packed, and the abdomen was then drained, or no attempt was made to remove the placenta with closure without drainage. The most satisfactory results were obtained in the cases where it was possible to remove the placenta completely. In several cases it was necessary to perform a supravaginal hysterectomy in removing the placenta.[3, 5, 7, 11]

CASE REPORT

N. W., aged 36 years, a Negro housewife, was first seen June 15, 1950, complaining of irregular menstruation for two months and moderate swelling of the lower extremities. She gave a history of one previous pregnancy, about ten years previously, which terminated spontaneously in the third month of pregnancy. On examination the uterus was found to be enlarged to the size of about a three months' pregnancy, with a small mass palpable above and to the right of the uterus. A diagnosis was made of ovarian cyst on the right, or fibroid uterus. Examination again on July 27, 1950, revealed the uterus to be larger, and positive breast changes. Although she continued to have irregular bleeding, a diagnosis was made of pregnancy, and the expected date of delivery was set for about Feb. 2, 1951. The prenatal course was uneventful except for continued menstruation at irregular intervals. She maintained a normal blood pressure, normal weight gain, and negative urine throughout. On Nov. 26, 1950, she was admitted to the hospital with the diagnosis of premature labor. Labor pains were excellent for over twenty-four hours without any dilation of the cervix. After about twenty-seven hours of continuous labor, it was decided to terminate the pregnancy by cesarean section. A low midline incision was made, under general anesthesia, and on opening the peritoneum a large amount of thin, flaky, yellowish fluid escaped. An incision was made in what was thought to be the uterus, but was later found to be the placenta. Bleeding was controlled with pressure until the female fetus was delivered with the aid of forceps. The baby, who weighed 5 pounds, cried immediately. The placenta was attached by a pedicle to the dome of the uterus and the right broad ligament. The placenta was removed entirely, with bleeding from the attachment sites controlled with transfixing sutures. The abdomen was closed without drainage. The

Received for publication, Aug. 3, 1953.

postoperative course was uneventful, and the mother and the baby were discharged from the hospital on the seventh postoperative day. The baby was normal in every respect. Follow-up in April, 1953, revealed the mother to be pregnant again, and the child developing normally.

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HERNIA THROUGH FORAMEN OF WINSLOW

Report of a Case With Preoperative Roentgen Diagnosis and Successful Surgical Management

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HERNIA through the foramen of Winslow is not a common occurrence. According to Lavarde and Chevret,[1] there have been less than seventy cases reported in the world literature, and of these, there were but six in which the preoperative diagnosis was suggested. Recently, Hollenberg[2] and Cimmino[3] recorded cases in which such a diagnosis was made. It is the purpose of this paper to present an unusual case of this rare internal hernia in which the diagnosis of herniation of the large intestine through the foramen of Winslow was made by roentgen studies and confirmed at operation.

ANATOMY AND PATHOLOGY

The foramen of Winslow or epiploic foramen is a potential canal connecting the general peritoneal cavity with the omental bursa. It is bounded anteriorly by the border of the lesser omentum with the common bile duct, hepatic artery, and portal vein. Posteriorly is the peritoneum overlying the inferior vena cava. Superiorly is the peritoneum covering the duodenum.[4] From a knowledge of these anatomic relationships, it can be seen that the surgical management must be accomplished with the utmost regard for the vital structures concerned.

Moynihan and Dobson[5] have postulated that one or more of the following conditions must be present for herniation to occur: (1) a common mesentery for the entire intestinal tract, (2) absence of secondary fusion of the ascending colon to the parietal abdominal wall, (3) an abnormally large foramen of Winslow, and (4) an abnormally long mesentery allowing undue mobility of the small bowel. Several authors including Cimmino have suggested that a large right lobe of the liver may also predispose to hernia in that a funnel-like approach to the foramen is thus formed.

SYMPTOMATOLOGY

Silverstone[6] has stated that cases of herniation may be either acute or long standing in nature. The acute cases are characteristically heralded by the signs and symptoms of intestinal obstruction, varying with the level at which obstruction has occurred. Usually this obstruction is precipitous in

Received for publication, Aug. 27, 1953.

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onset, although occasionally symptoms may be present for some days before the patient consults a physician. Infrequently, however, herniation may be present for a long time before actual obstruction intervenes. Indeed, Dewis[7] presented a case in which the history and roentgen findings suggested that herniation had been present for some seventeen years prior to complete obstruction and sudden death.

CASE REPORT

Mrs. A. W., a 54-year-old white woman, was admitted to the Graduate Hospital Medical Service on Nov. 18, 1952, complaining of epigastric fullness, pain in the right upper quadrant, and intermittent vomiting. Her past history was of interest in that for the previous five years she had been under treatment for giant follicle lymphoblastoma, for which she had received both roentgen and nitrogen mustard therapy.

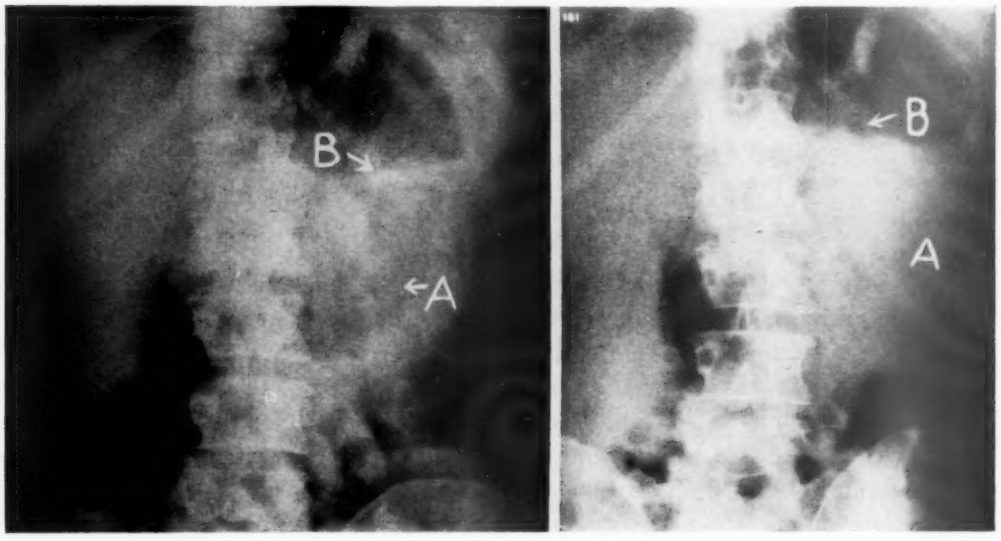


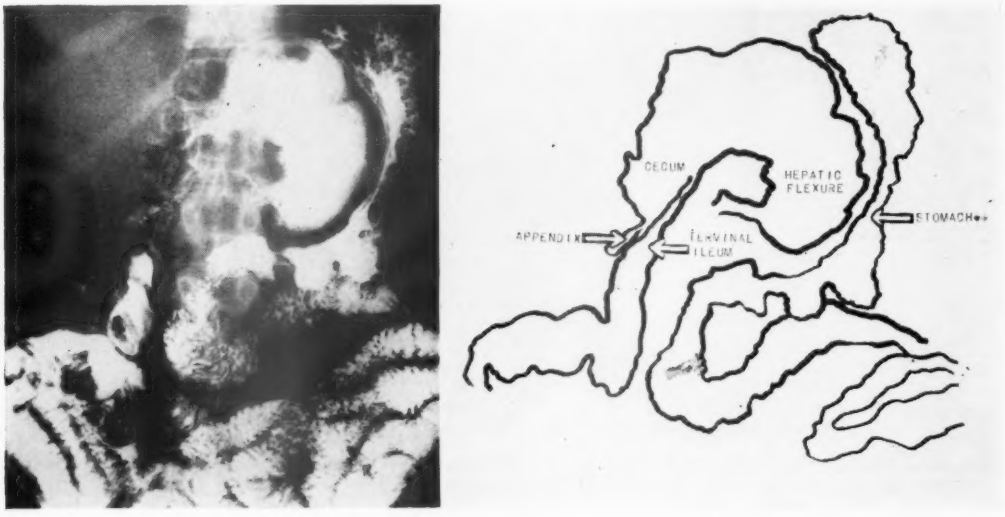
Fig. 1.—Anteroposterior recumbent (*left*) and erect (*right*) films of abdomen on admission. Large collection of gas (*B*) to left of midline just below diaphragm exhibits fluid-air level in erect position. This lies medial to stomach (*A*) and represents the cecum and ascending colon herniated into the lesser omental sac.

The patient dated the onset of the present difficulties to three weeks prior to admission, when for the first time she noticed a sense of upper abdominal fullness following each meal. This sensation would persist for several hours, with slight relief being obtained by the ingestion of large quantities of water. Gradually, the patient had restricted her diet entirely to fluids. One week prior to admission she began to have occasional vomiting. She denied hematemesis. Associated with the vomiting was an increasingly severe "griping" pain beneath the left anterior costal margin. On Nov. 16, 1952, the pain was more constant, requiring injection of morphine by her physician. On this date she had a small, formed bowel movement, but no further passage of flatus or feces was noted until admission.

Physical examination revealed a moderately dehydrated patient lying quietly in bed, not appearing in any acute distress. Temperature was 100° F. rectally, pulse 90 per minute, blood pressure 144 mm. Hg systolic and 80 mm. Hg diastolic. The primary findings were confined to the abdomen, which was slightly protruberant but quite soft to examination. The liver was palpable to a point slightly below the level of the umbilicus. A globular, solid mass was present in the epigastrium and left upper quadrant which was interpreted as spleen. No tenderness was elicited; by auscultation, peristalsis was not obstructive. The rectum was empty of feces.

Laboratory data on admission were reported as: hemoglobin 12.1 Gm., white blood cells 5,000 cells per millimeter with a normal differential, blood sugar 103 mg. per 100 ml., blood urea nitrogen 8 mg. per 100 ml. Electrolytes were all within normal limits; the urine presented no albumen or sugar. Liver function tests were normal.

On the day following admission, survey films of the abdomen made in the anteroposterior recumbent and erect positions (Fig. 1) failed to demonstrate any evidence of intestinal obstruction. An unusual, large radiolucent shadow which exhibited an air-fluid level in the erect position was noted medial to the stomach. This evidently represented the cecum and ascending colon in the lesser peritoneal sac, but its significance was not appreciated at this time. Because of the inability to make a definitive diagnosis from the survey films, and since there was no roentgen or clinical evidence of obstruction, it was recommended that the patient be given a barium mouth meal.



A. *B.*

Fig. 2.—Anteroposterior film (*A*) following barium meal with schematic drawing (*B*) showing stomach and entire small intestine filled with barium. Large collection of barium medial to stomach is in the herniated cecum and ascending colon.

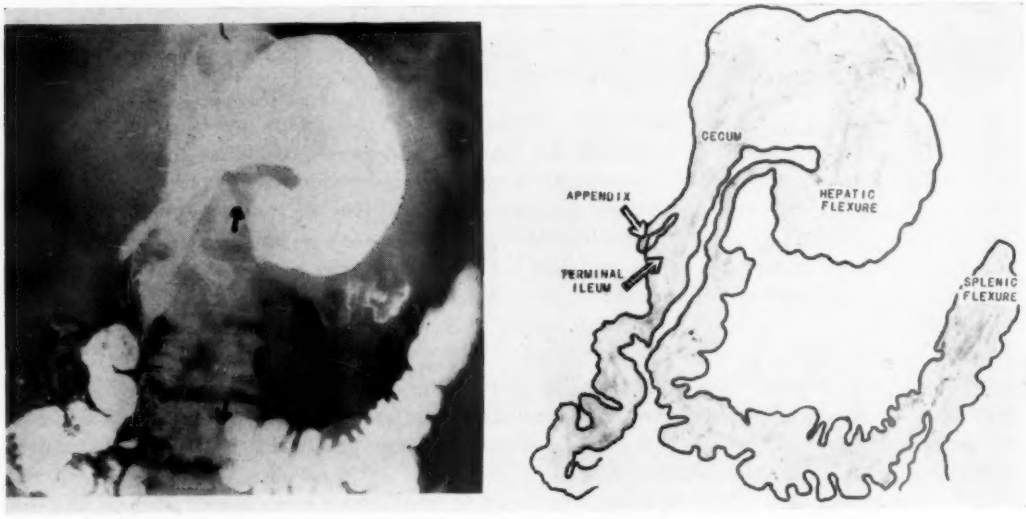
The next day (Nov. 20, 1953) the patient was given six ounces of a barium-water mixture by mouth. In fifteen minutes the barium had passed through the jejunum and most of the ileum; the head of the column was directed cephalad and medially, where the barium seemed to enter a pouch located medial and posterior to the stomach. Shortly after this, when the appendix filled with barium, it became apparent that this pouch represented the cecum, ascending colon, and the hepatic flexure, which were herniated into the lesser sac (Figs. 2 and 3). In order to determine accurately how much of the colon was involved in the hernia, a small barium enema was then given. Almost complete obstruction to the flow of barium was encountered in the proximal transverse colon (Fig. 4).

On the evening of this examination, the patient complained of cramping, upper abdominal pain, but had no vomiting, temperature, or pulse elevation and was passing flatus by rectum. Physical examination was unchanged and peristalsis was not obstructive. However, on the morning of Nov. 22, 1953, the pain began to increase in frequency and severity. Present in the epigastrium was a tympanitic, fluctuant, tender mass. The abdomen was generally distended, and peristalsis was high pitched and obstructive in nature. Temperature was 99° F. orally, pulse 120 per minute. Clinically, the patient appeared acutely ill. A survey film of the abdomen at this time revealed a large amount of barium in the herniated proximal colon in the same position as demonstrated on previous films.

No barium was seen in the distal half of the colon. There was no evidence of small intestinal obstruction.

A surgical consultation was requested. The roentgen diagnosis of hernia of the large bowel into the lesser sac was concurred, and immediate exploration was decided upon. On opening the abdomen through a long upper midline incision, there was release of about 500 c.c. of clear, odorless, straw-colored fluid from the peritoneal cavity. The right lobe of the liver was greatly enlarged, extending into the right lower quadrant and presenting

Fig. 3.—Lateral view showing position of hernia. White dotted line represents dome of diaphragm.



A. *B.*

Fig. 4.—Anteroposterior recumbent film (*A*) and schematic drawing (*B*) following combined barium meal and cautious barium enema, demonstrating amount of colon in the hernia and partial obstruction in proximal transverse colon indicated by black arrows.

a very hard mulberry-like appearance suggestive of far-advanced cirrhosis. A wedge biopsy was taken of this lobe. The spleen was enlarged to about three times normal size; it was soft and mushy to palpation. Immediately below the incision was a large, distended, markedly inflamed, tense loop of large bowel. With further exploration, it was evident

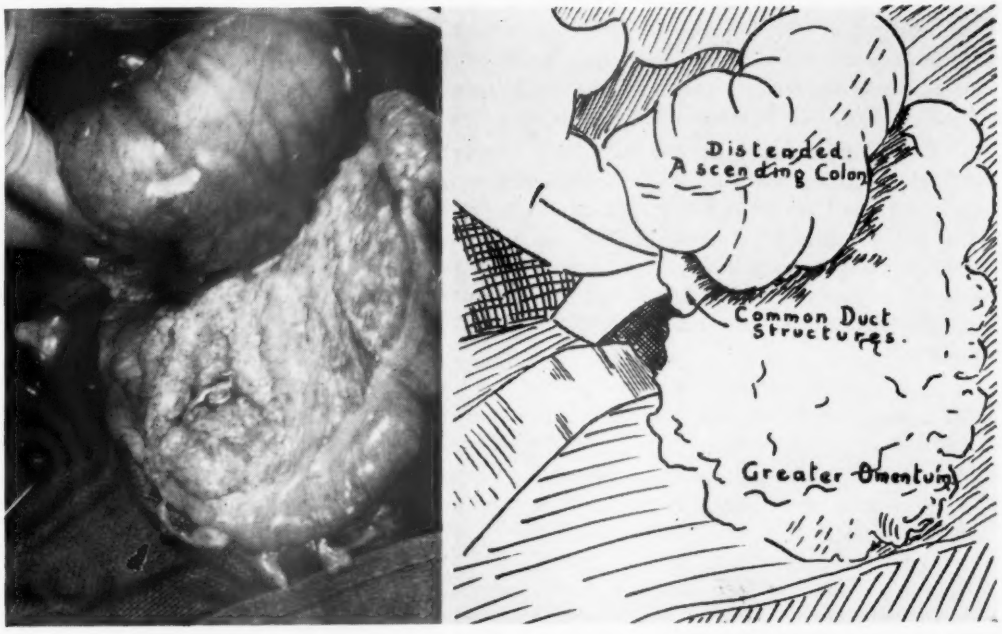


Fig. 5.—Photograph and accompanying diagram demonstrating the appearance of the herniated bowel as it presented through the tear in the gastrohepatic omentum.

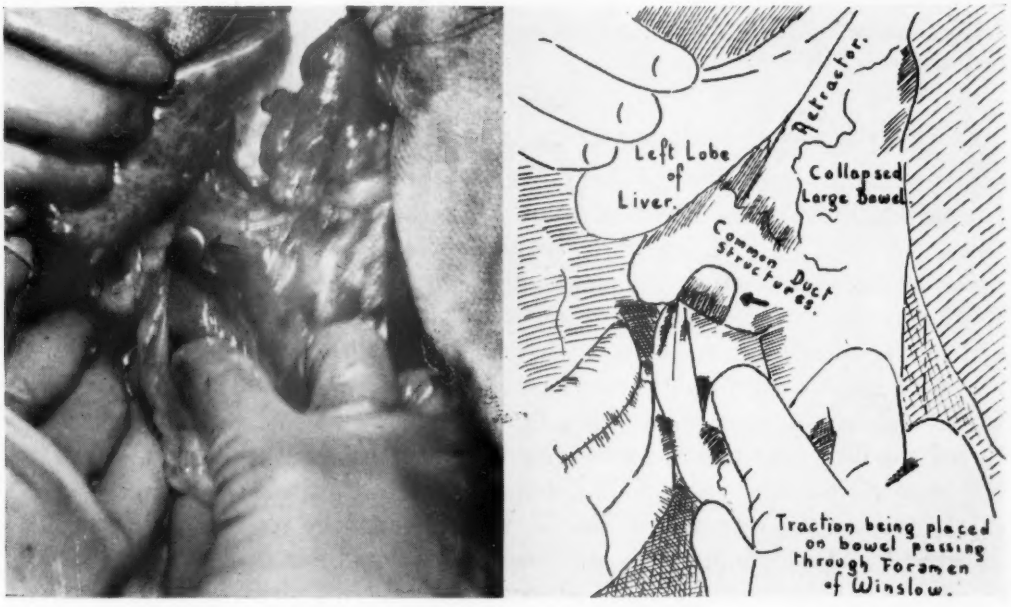


Fig. 6.—Appearance of bowel following aseptic enterotomy as traction is being exerted to reduce through the foramen of Winslow. A retractor is seen elevating the common duct structures. Arrow indicates retractor in the foramen of Winslow.

that this represented the entire ascending colon to a point just distal to the hepatic flexure, which was presenting through a large rent in the gastrohepatic omentum (Fig. 5). The mode of entry was apparent with the finding of a slightly distended loop of terminal ileum and collapsed transverse colon tightly compressed at the foramen of Winslow. Efforts to reduce the hernia by gentle manipulation were unsuccessful; an aseptic enterotomy was performed and severeal hundred cubic centimeters of thick semisolid barium was aspirated, with collapse of the involved bowel. Following this, the enterotomy was closed. At the completion of this procedure, it was possible to reduce the bowel through the gastrohepatic omentum with gentle traction and thence through the foramen of Winslow without difficulty (Fig. 6). Within a short time, the bowel appeared viable.

Two fingers could be slipped easily through the foramen of Winslow. The rent in the gastrohepatic omentum was closed with interrupted sutures of cotton, following which two interrupted sutures of the same material were used to suture the periductal fat loosely along the common duct structures to the peritoneum overlying the inferior vena cava in an effort to obliterate the portal through which herniation had occurred. The ascending colon was then returned to its normal position in the right lower quadrant, and several fat tabs sutured to the lateral peritoneal wall to hold it in place. The abdomen was closed.

The immediate postoperative period was uneventful. However, forty-eight hours after operation the abdomen became quite distended, and it was apparent that there was definite fluid in the flanks. The liver biopsy taken at the time of surgery was reported to represent "far-advanced portal cirrhosis." Twenty-four hours later, spontaneous drainage of clear ascitic fluid began to occur from the lower angle of the incision, which continued intermittently for several days. Nine days postoperatively, all liver function tests were normal with the exception of a Bromsulphalein retention of 27.3 per cent However, clinically the patient did very well and there was no further evidence of intestinal obstruction. She was placed on a liver protection program of dietary supplement and parenteral proteins, and subsequently transferred to the Gastrointestinal Service for further treatment. A roentgen examination of the intestinal tract on Jan. 15, 1953, revealed no evidence of abnormality.

However, the cirrhosis failed to respond to the most intensive therapy, and three months following surgery (Feb. 9, 1953) she rapidly went into congestive heart failure and expired.

DISCUSSION

All of the roentgen signs of herniation through the foramen of Winslow concern themselves with the demonstration of air in the lesser sac. Case and Upson[8] stated that the demonstration of small intestine anterior to the hepatic flexure or first part of the transverse colon is necessary to establish the diagnosis. This sign is obviously not applicable when these portions of the colon are herniated in association with the small bowel, nor as in this case in which the small bowel did not enter the hernia. Hollenberg[2] emphasized the importance of gas in the lesser omental sac in association with obstruction in the transverse colon as demonstrated by barium enema. He made what he believed the first preoperative roentgen diagnosis of lesser sac hernia. Cimmino[9] has pointed out the roentgen findings in perforation into the lesser sac. In a later report[3] he described a case in which gas in the lesser sac due to hernia differed from the gas noted in perforation. We believe that once the presence of gas in the lesser sac can be determined with certainty, then the diagnosis rests between a perforation into the lesser sac or a hernia. We agree with Cimmino that if the fluid levels do not conform to the anatomic recesses of

the sac or if the actual wall of the bowel can be demonstrated, then the specific diagnosis of herniation into the lesser sac can be made without subjecting the patient to barium studies.

SUMMARY

1 A case of herniation of the ascending colon and portion of the transverse colon through the foramen of Winslow which was diagnosed preoperatively and successfully treated surgically is presented.

2. The roentgen findings are discussed.

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BENIGN STRICTURE OF HEPATICODUODENOSTOMY: ITS TREATMENT

Report of Two Cases

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THE procedure described in this report has been successfully employed in the treatment of two cases of benign stricture of hepaticoduodenostomy. The following two cases are reported.—

Case 1.—A 49-year-old white woman in 1931 had a cholecystectomy for cholecystitis and cholelithiasis elsewhere. During this operation an accidental excision of the extrahepatic biliary tract was made. At the second operation, no part of the hepatic and common bile ducts was found. This defect was repaired by anastomosing the adjacent duodenum to an opening in the liver at the hilus, through which much "white bile" was obtained. A 10 cm. piece of 24 Fr. rubber catheter, with a cuff, was used as a splint. Five months later this splint was removed by transpyloric forceps manipulation through a gastrotomy.

The patient remained well for six years (1937) when she developed evidence of gradually increasing obstruction of the biliary tract. Surgery was postponed too long chiefly because of the realization of the difficulties of exposing the site of obstruction. When finally relief became imperative, the liver was so large that exposure of the anastomosis at the hilus was technically near impossible.

Operation: A right upper rectus incision was used. The second portion of the duodenum was mobilized for 8 cm. The mobilized duodenum was opened longitudinally so as to expose the previous hepaticoduodenostomy. The stoma would just admit a 1 mm. probe. This opening was dilated to 30 Fr., using urethral sounds. Exploration of the intrahepatic ducts by probe, scoops, and catheter irrigation revealed twenty-five soft calculi up to 1 cm. in diameter. These calculi were removed. A 15 cm. piece of 24 Fr. rubber catheter, with a cuff, was inserted about 6 cm. up into the liver through the dilated stoma. The opening in the duodenum was closed transversely. The abdomen was closed in layers with one Penrose drain. The postoperative course was uneventful, and recovery from the obstructive jaundice was rapid.

During the past sixteen years the patient has been examined at least once a year and has been in good health. The liver has remained above the right costal margin. The patient has gained 25 pounds in weight and has been free of jaundice. In 1939 she experienced several episodes of chilliness, nausea, and epigastric pain during a two-week period. This patient was last seen on March 17, 1953.

Case 2.—In 1946, Mrs. L. H. had a cholecystectomy performed elsewhere. During this operation an accidental excision of the hepatic duct, from just at the junction of the right and left hepatic ducts, and the common bile duct was made. Approximately one month later, this was repaired by a hepaticoduodenostomy. Following an uneventful convalescence the patient continued well until 1948, when evidence of benign recurrent obstruction of the biliary tract developed. The patient was prepared for surgery.

Operation: Through a right upper rectus incision, an enlarged liver was revealed. The duodenum was found attached high up into the hilus of the liver and was rather easily

Received for publication, Sept. 1, 1953.

mobilized in spite of the adhesions and the huge liver. The duodenum was opened longitudinally opposite the anastomosis. The hepaticoduodenostomy stoma, narrowed to 1 mm., was readily located with a fine probe. It was dilated to 26 Fr., using common duct dilators. When this opening was dilated, a considerable amount of turbid bile, containing many soft calculi up to 3 mm. in diameter, poured out. A section of 24 Fr. rubber catheter, with a cuff, was introduced high into the intrahepatic ducts. Approximately six inches of the catheter extended down into the duodenum. The opening in the duodenum was closed transversely. A Penrose drain was inserted down to the operative area.

The postoperative course was uneventful, with an immediate abatement of symptoms. The patient has continued in good health to the present time.

DISCUSSION

The technique described in this report offers a rational, simplified surgical approach to a definite, although limited, group of cases of biliary obstruction in adults.

Walters and Phillips,[1] reporting on 165 patients with strictures of the extrahepatic bile ducts, listed forty-two cases of hepaticoduodenostomy with 52 per cent recurrence of biliary obstruction.

Walters and Kelly,[2] reporting on an additional 100 cases from 1948 through 1951, listed forty-six cases of anastomosis of hepatic duct to duodenum, of which thirty-two cases were traced with a recurrence of stricture in 34.4 per cent.

Lahey and Pyrtek,[3] in discussing the operative management of 280 strictures of the bile ducts, reported that of forty-five patients with biliary-intestinal anastomosis (jejunum and duodenum) from 1940 to 1948, eleven developed recurrence of biliary obstruction.

Cole, Reynolds, and Ireneus[4] have stated, "The tendency to recurrence of stenosis in patients without any duct at the hilus has become the greatest single obstacle to a successful outcome, and attempts to prevent this stenosis have represented the major part of all recent surgical effort in this field."

In the past, reconstructive or plastic types of operation upon the stricture have been the common practice.[5-7]

The seriousness of these recurrent benign strictures of the common hepatic duct is realized since Longmire and Sanford[8, 9] have recommended intrahepatic cholangiojejunostomy with partial hepatectomy for their treatment.

Borrowing a principle from the urologist, namely, that an acquired benign stricture of a mucosa-lined tube can be dilated and will remain patent, it was applied to these cases of benign stricture of a hepaticoduodenostomy with excellent results in both cases.

The potential complications which may be anticipated are that a duodenal fistula may develop or the stoma may not be found due to marked narrowing or complete obliteration. These complications have not occurred in the reported cases. The risk of development of a duodenal fistula is no greater in this operation than in the usual reconstructive or plastic type of procedure. Certain advantages are apparent, but will be mentioned for completeness. In this operation, certain important anatomic structures, namely, the portal vein,

hepatic artery, and inferior vena cava, are avoided by opening into the duodenum and making a transduodenal dilation of the stricture.

Technically, this operation is less difficult. It may be performed by the less skilled surgeon. The facility with which the pathology in these cases was corrected is evident from the two reported cases. In comparison with the arduous reconstructive or plastic type of operation, this procedure involves less risk for the severely ill patient.

Another suggested advantage is that this operation may be utilized in those patients who are beginning to show evidence of recurrence of a stricture, whereas one would hesitate to perform the greater-risk reconstructive or plastic-type operation at this time By having this lower-risk operation available, the patient can be treated earlier and is in better condition for surgery.

This procedure should be of value in benign stricture of choledochoduodenostomy and choledochojejunostomy.

The results obtained in these two cases have been very satisfactory. One patient has been asymptomatic and in good health for five years. The other patient has remained in good health for sixteen years with the exception of one two-week period of recurrent episodes of epigastric pain, nausea, and chilliness.

SUMMARY

1. A low-risk operation for the treatment of benign stricture of a hepaticoduodenostomy is presented.

2. This operation has been used in the two reported cases with satisfactory results.

3. The possible complications and advantages are discussed.

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SPONTANEOUS RUPTURE OF THE NORMAL URINARY BLADDER

Report of One Case

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SPONTANEOUS rupture of the urinary bladder is defined as rupture of the bladder without antecedent trauma. The purpose of this paper is to add an interesting case report to the literature of this relatively rare entity. The first comprehensive report on spontaneous rupture of the urinary bladder was that of Stone[1] in 1931, who collected forty cases in the literature up to that time and added two cases of his own. Since then, nineteen more cases have been recorded and reviewed by Crastnopol[2] in 1950.

Spontaneous rupture of the bladder is usually associated with (1) inflammation of the bladder wall, (2) malignant disease of the bladder, or (3) overdistention of the urinary bladder secondary to obstruction or due to alcohol depression of the sensorium. Spontaneous rupture of a normal bladder is rare. Barnes and Steele[3] reported the occurrence of spontaneous rupture in a normal bladder.

Prompt diagnosis of rupture of the bladder and institution of proper therapy is imperative in order to keep the mortality rate from this condition at a minimum. Diagnosis is usually difficult because of the rarity of this entity and because the patient usually presents the signs and symptoms of an acute intra-abdominal process. Therapy in the following case was delayed because of the difficulty in establishing a diagnosis and correlating the urologic radiographic findings with the clinical condition of the patient. The working diagnosis in this case was a perforated peptic ulcer. A diagnostic paracentesis demonstrated spermatozoa which directed our attention to the urologic tract.

CASE REPORT

A Negro male chef, aged 61 years, entered our Emergency Ward at 1:30 P.M. on March 11, 1953, with the chief complaint of severe generalized abdominal pain. The history was obtained from the patient and his wife. It was stated that the patient had had intermittent episodes of epigastric nonradiating pain for many years. The pain occurred usually between meals and occasionally woke the patient up at night. The pain was relieved by the ingestion of food and medication given by his local physician. The patient had been told that he had a "nervous stomach." The patient stated that he had been able to maintain his daily work and got along fairly well except for the three months prior to admission, when he had onset of increasing weakness, and he had noted a loss of fifteen pounds during this period. One week prior to admission the patient had onset of severe epigastric pain and generalized malaise. He stayed home from work and at bed rest. The symptoms of malaise persisted with intermittent epigastric pain until 5:30 A.M. on the morning of admission, when he was awakened from his sleep with severe epigastric pain associated with retching without the production of much vomitus. The patient stated

Received for publication, Sept. 8, 1953.

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that the retching was quite severe. During the episode of retching the patient noted the exaggeration of the epigastric pain, which was more severe than anything he had noted before. The epigastric pain subsided, and then the patient began to complain of generalized abdominal pain predominantly in both lower quadrants. He had a normal bowel movement twenty-four hours before his admission and had passed flatus on the morning of admission.

There were no genitourinary symptoms. Past history revealed that the patient had an appendectomy and right herniorraphy in 1932, and he had had gonorrhea and syphilis in 1918. He was treated with arsphenamine for the latter. There was no history of tuberculosis, diabetes mellitus, or neurological disorders.

Physical examination revealed a thin, anxious Negro male who was in acute distress and appeared slightly disoriented. Pulse was 100 and regular, respirations were 22, blood pressure 186/90, and the temperature was 100° F. rectally. Both pupils were fixed. Examination of the abdomen revealed a slightly distended abdomen with an absence of bowel sounds. The abdomen was generally rigid and approached a boardlike abdomen, with marked tenderness in the right lower quadrant. Rectal examination was negative. Neurological examintion was entirely negtive except for the fixed pupils.

Admission laboratory studies revealed a hemoglobin of 16.6 Gm., a white blood cell count of 15,600, nonprotein nitrogen was 81, bilirubin was 0.6, CO_2 was 46 volumes per cent, chloride was 102 meq. per liter, potassium was 5.5 meq. per liter, sodium was 147 meq. per liter, and serum amylase was 95. A catheterized urine specimen was yellow in color, had 3+ turbidity, a reaction of 4.5, a specific gravity of 1.021, albumin, sugar, acetone, and bile were absent and there were 0 to 2 white blood cells per high-power field and 100 red blood cells per high-power field. A chest film demonstrated plate atelectasis on the right. Recumbent and upright films of the abdomen revealed some gas-filled, slightly dilated small intestinal loops and gas in the colon. The roentgenographic impression was that this represented an acute abdomen, possibly secondary to peritonitis with probable atony of the small intestine. A possible diagnosis of perforated peptic ulcer was made, and the patient was watched carefully. Eight hours after admission the patient's temperature, pulse, respirations, and x-ray film of the abdomen were the same as on admission. The patient still had a semirigid abdomen, but he was in only moderate distress. The white blood cell count had not changed and the urinalysis was essentially the same except that it had 20 to 30 red blood cells per high-power field.

A diagnostic paracentesis was performed in the right gutter using a No. 18 needle and 50 c.c. syringe. The paracentesis was performed in the right lower quadrant 4 cm. medial from the superior iliac spine. The bladder was first evacuated by catheterization. The paracentesis produced 3 L. of a thin, smoky fluid. Immediately after withdrawal of this fluid the patient became much more comfortable, the abdomen became softer instantaneously, and there was marked subjective relief of pain. The fluid had a uriniferous odor and had a pH of 7.5. It contained 100 red blood cells per high-power field and 5 to 10 spermatozoa per high-power field. After the finding of the spermatozoa in the paracentesis fluid, the patient was again immediately catheterized to determine the character of the bladder urine. The bladder urine was yellow in color and had a pH of 4.5. The working diagnosis was now changed to intra-abdominal perforation of a large bladder diverticulum with a ball-valve type of opening. The patient was placed on constant drainage. He was now completely asymptomatic, and the abdominal findings had disappeared. The vital signs remained stable, as did the white blood cell count throughout the first hospital day.

The morning of the second hospital day the patient was still asymptomatic and an intravenous pyelogram revealed a normal upper urinary tract and bladder. There was no evidence of perforation of the bladder or of diverticulum of the bladder. A cystogram was performed by filling the bladder with sodium iodide through a urethral catheter. During the time the cystogram was being taken the patient complained of no pain in the abdomen and there was no change in the abdominal findings. Not all the sodium iodide that was placed into the bladder returned through the catheter. The cystogram revealed a

smoothly outlined normal bladder with a saccular pouch formation behind it that was interpreted as being a large diverticulum or possibly an intraperitoneal rupture of the bladder. The patient continued to be asymptomatic and the abdominal findings were negative, and although the possibility of an intra-abdominal rupture of the urinary bladder was considered, the abdominal findings following the paracentesis did not correlate with this diagnosis. It was decided to observe the patient further in order to establish a more positive diagnosis.

The following day the patient had a cystoscopic examination and another cystogram, and a definite diagnosis of intraperitoneal rupture of the bladder was made. On the posterior wall of the bladder there was a small hole, the edges of which were normal and did not seem inflamed. The cystogram definitely revealed an intraperitoneal rupture of the bladder. During the cystoscopy the patient had no discomfort and there was no change in the abdominal findings. All the fluid that was introduced into the bladder, however, did not return. The patient was immediately prepared for surgery and an exploratory laparotomy revealed a normal urinary bladder without signs of inflammation or tumor. There was no bladder neck obstruction. There was a small opening in the posterior aspect of the dome of the bladder immediately above the trigone that connected with the intraperitoneal cavity. The area around the pin-point opening was slightly injected. Tissue taken from this area for biopsy was reported as normal bladder tissue with some hemorrhage in it. Although the intraperitoneal cavity contained some free fluid, the abdomen was remarkably free of any signs of inflammation. The area of rupture was sutured and the patient was placed on cystotomy and urethral drainage. The patient was treated with penicillin and streptomycin postoperatively. His course after surgery was entirely benign. The nonprotein nitrogen returned rapidly to normal, and the patient's urine became free of red blood cells. Evaluation of the patient's syphilitic state revealed a negative serum Hinton serologic test, but the cerebrospinal fluid serology was positive with a paretic colloidal gold curve. On the nineteenth postoperative day the patient had a repeat cystogram and cystometry study, which were both normal.

DISCUSSION

This represents a case of spontaneous rupture of a normal urinary bladder. Although spontaneous rupture of the urinary bladder associated with tumors, infections, or obstruction is rare, the occurrence of rupture in a normal bladder is rarer still, and without explanation.

It is postulated that this patient's bladder had been perforated intraperitoneally for sixteen hours before the paracentesis was performed and he was placed on constant drainage. This duration of the perforation was long enough for the patient to dialyze his own urine to a neutral pH and to produce a state of acute uremia. Although the bladder had been perforated for this protracted period, the patient's vital signs were normal and the abdomen was free of any signs of peritonitis at the time of operation. Immediately upon evacuation of the abdominal fluid on the night of admission, the patient's abdominal pain and rigidity disappeared.

The high mortality rate reported in the literature is due to (1) the preponderance of cases in the preantibiotic era and (2) the fact that the correct diagnosis was not made in many cases. It was fortuitous in this case that the patient's urine was sterile despite the lack of antibiotic therapy preoperatively. Cultures of the urine and the paracentesis fluid were negative.

Diagnosis and therapy in this case were delayed, paradoxically, not because the correct entity was not considered but because the patient's good clinical

condition did not correlate with the diagnosis of intraperitoneal rupture of the urinary bladder. Although the first cystogram indicated the possibility of an intraperitoneal rupture of the bladder, the patient had no abdominal pain or signs of peritoneal irritation while the cystogram was being taken. The diagnosis was finally established when the perforation was visualized cystoscopically.

Intensive investigation of the patient's home life and social background rules out the possibility of trauma as the etiology of the bladder rupture. Direct visualization and biopsy of the bladder rules out obstruction, tumor, or inflammation as the cause of the rupture. Despite the finding of a positive cerebrospinal fluid Hinton test, the only syphilitic neurological manifestations were fixed pupils. Cystoscopy and cystometry ruled out the possibility of a neurogenic bladder secondary to syphilis.

Most cases of spontaneous rupture of the urinary bladder are misdiagnosed as rupture of an intra-abdominal viscus. This was the primary diagnosis on this patient because of his past "ulcerlike" history. Evaluation of the patient's gastrointestinal tract did not demonstrate the cause of the gastrointestinal complaints.

It is interesting to speculate as to whether the patient's retching without the production of vomitus created increased abdominal pressure of such a magnitude as to cause a rupture of a full normal urinary bladder. A review of the literature did not reveal a case report of any rupture of the bladder secondary to increased abdominal pressure produced by the physiologic process of vomiting.

SUMMARY

1. A case report of spontaneous rupture of a normal urinary bladder is presented.

2. The possibility of the effect of increased abdominal pressure due to vomiting on a full urinary bladder as an etiological factor is postulated.

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CONGENITAL EVENTRATION OF THE DIAPHRAGM IN INFANCY

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EVENTRATION of the diaphragm is defined as an abnormally high position of one leaf of the intact diaphragm. From a study of the etiology of this condition, Beck[1] concluded that all the evidence points to the fact that eventration of the diaphragm is a congenital lesion. The true incidence of congenital eventration of the diaphragm can be determined only by the routine use of roentgenologic studies of the newborn child. In this manner, Beck and Motsay[2] found that about 4 per cent of 2,500 newborn children had some diaphragmatic weakness, but in only three was the condition productive of severe respiratory difficulty.

The feasibility of surgical correction of congenital eventration of the diaphragm in infancy was first demonstrated by Bisgard[3] in 1947, who reported an operative cure in an infant 6 weeks old. Subsequently, State[4] Butsch and Leahy,[5] and Shellito and Bartlett[6] each reported an operative cure in an infant with this condition. In a group of fifteen infants requiring surgery for congenital anomalies of the diaphragm, Koop and Johnson[7] reported three cures in infants with eventration.

The following case is that of the youngest patient yet reported with congenital eventration of the diaphragm treated successfully by surgical repair.

CASE REPORT

History.—L. G., a white female infant aged 8 days, was admitted to the Mount Sinai Hospital on July 3, 1952. The patient had been born at full term, by forceps delivery. Pregnancy was uncomplicated except for some fever at three months. The family history was irrelevant. Dyspnea and cyanosis had been noted since the fifth day of life. The respiratory difficulty increased, requiring inhalations of oxygen.

Physical Examination.—The infant was well developed and well nourished, weighing 7 pounds, 4 ounces. The temperature was 99.6° F., the pulse rate 120, and the respiratory rate 58. Some dyspnea and cyanosis were noted. There were no abnormalities of the head, extremities, abdomen, or genitals. Examination of the chest revealed dullness to percussion and diminished breath sounds at the left base. The heart sounds were maximal in the right chest.

The hemoglobin content was 20 Gm.; the leukocyte count was 5,000, with polymorphonuclear leukocytes 51 per cent, lymphocytes 45 per cent, monocytes 2 per cent, and eosinophils 2 per cent. Urinalysis was normal.

Roentgenograms of the chest and abdomen revealed the left diaphragm to be elevated to the level of the third rib posteriorly; loops of colon filled with radiopaque material were noted in the left chest below the level of the diaphragm (barium had been administered prior to admission); the heart was markedly displaced to the right side (Fig. 1). Fluoroscopic examination revealed slight respiratory motion of the left diaphragm.

Operation was performed on the day of admission. Under ether anesthesia, a left oblique subcostal incision was made. The stomach and loops of small and large intestine

Received for publication, Sept. 8, 1953.

were visible in the abdomen. The left lobe of the liver, the spleen, and the splenic flexure of the colon were located in the thorax. The left leaf of the diaphragm was high in the chest, and was represented by a thin translucent membrane, through which the pericardium and the left lower lobe of the lung were visible. The anterior edge of the diaphragm at its attachment to the chest wall showed a thin cuff of muscle. There were no muscular components in the remainder of the diaphragm, which continued as a thin membrane to its attachments to the lateral costal margin and the crura. The esophageal hiatus was visualized, and presented no abnormalities.

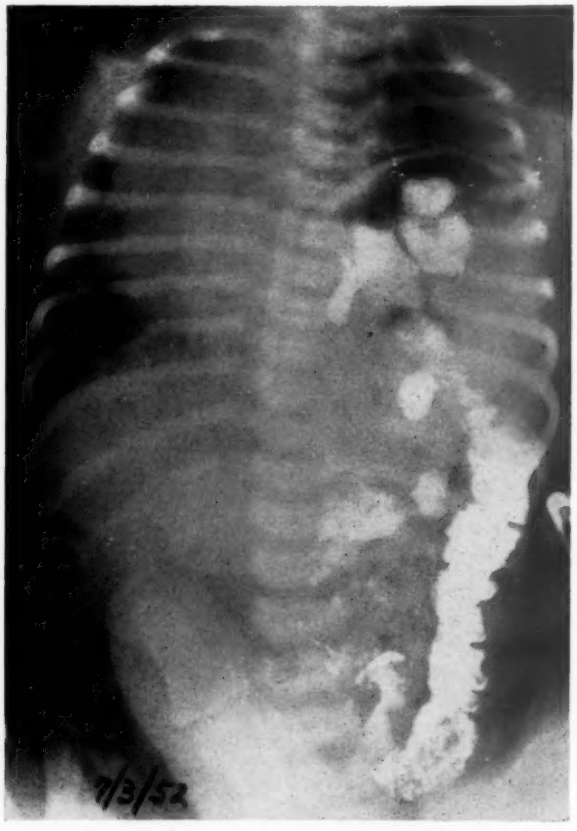

Fig. 1.—Roentgenogram of the chest and abdomen showing elevation of the left diaphragm, barium-filled splenic flexure of the colon beneath it, and displacement of the heart to the right side.

The left lobe of the liver, the spleen, and the splenic flexure of the colon were mobilized and brought down to the level of the abdominal wound. Babcock forceps were applied to the atrophic diaphragm, which was withdrawn from the chest into the abdomen The diaphragm was reconstructed by using plication mattress sutures of silk applied so as to bring various layers of the diaphragm in approximation with each other, and at the same time lowering the position of the diaphragm. The margin of the diaphragm was then sutured to the thoracic wall and renal fascia. The abdominal wall was closed with sutures of fine steel wire.

Pathologic Report.—A specimen of diaphragm was composed mainly of fibrous tissue covered on both surfaces by mesothelial cells.

Course.—An intravenous infusion of 5 per cent dextrose in saline solution and 100 c.c. of citrated blood was given during the operation. The infant was placed in an oxygen

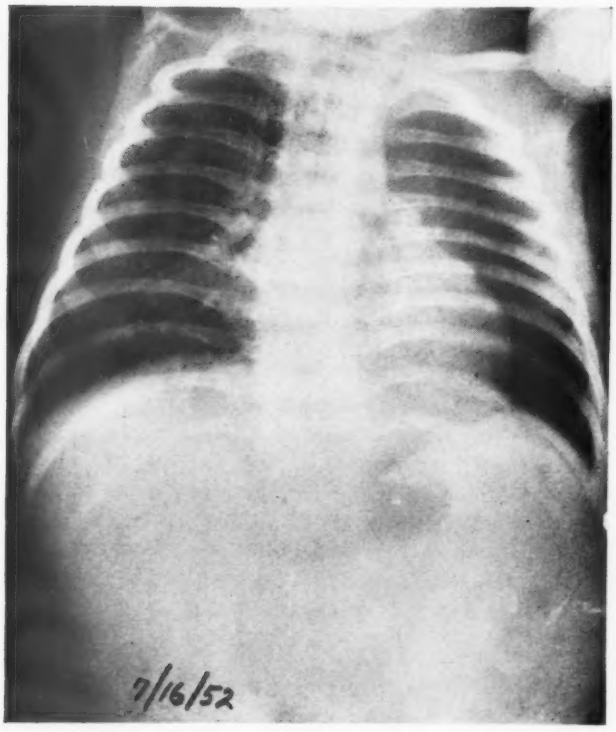

Fig. 2.—Roentgenogram of the chest taken thirteen days after the operation showing complete aeration of the lungs, and normal position of the diaphragm and heart.

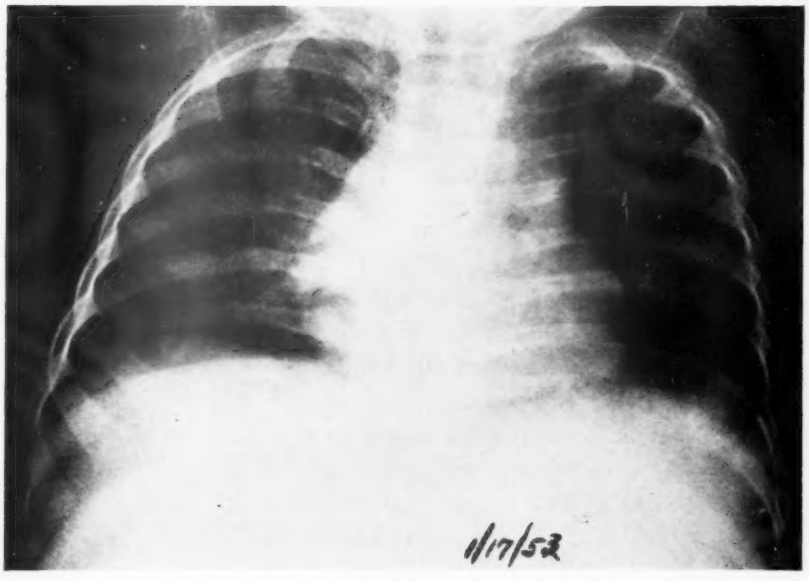

Fig. 3.—Roentgenogram of the chest taken six months after the operation, showing normal appearance of the diaphragm, heart, and lungs.

cubicle, the indwelling Levin tube was attached to a suction apparatus, parenteral fluids were continued, and penicillin was started.

The temperature remained normal throughout the postoperative course. Upon returning from the operating room the pulse was of good quality and the respiratory rate 50. There was no respiratory distress or cyanosis. Examination of the chest revealed slight dullness on percussion and diminished breath sounds in the lower part of the left side. The heart was in its normal position. During the first postoperative day, feedings were well tolerated, and there were normal bowel movements. Gastric siphonage and administration of intravenous fluids and oxygen were discontinued. Examination of the chest revealed no abnormalities on the second day. A roentgenogram of the chest, on the fifth day, showed good aeration of the lungs, a small effusion in the left costophrenic sinus, and normal position of the diaphragm and heart. Fluoroscopic examination revealed slightly diminished excursion of the left leaf of the diaphragm. The wound healed by primary union. Penicillin was discontinued on the seventh day; a total of 2.4 million units had been administered. A roentgenogram of the chest, on the thirteenth day, showed no abnormalities (Fig. 2). The infant was discharged in excellent condition on the fourteenth postoperative day, weighing 7 pounds, 9 ounces.

At follow-up examination six months after operation, the weight was 15 pounds and the height twenty-six inches. There were no complaints, and no abnormalities were noted on physical examination. A roentgenogram of the chest revealed normal position of both sides of the diaphragm; the heart and lungs appeared normal (Fig. 3). Fluoroscopic examination of the chest showed slightly diminished excursion of the left side of the diaphragm. Subsequent examination, one year after operation, revealed continued growth and development, the weight being 23 pounds, 4 ounces and the height thirty-two inches. A roentgenogram of the chest was normal.

DISCUSSION

The surgical management of congenital eventration of the diaphragm in infancy has rarely been the subject of published reports (seven cases in the literature[3-7]). Experience with one case prompted a review of the literature, and an analysis of these cases will be presented.

The age of the patients at operation varied from 8 days to 13 months as follows: 8 to 9 days in two, 6 weeks to 5 months in five, and 13 months in one.

There were six boys and two girls.

The symptoms of congenital eventration of the diaphragm in infancy were mainly respiratory, with varying degrees of dyspnea and cyanosis. These were either constantly present or made their appearance only when the respiratory reserve was needed, as during crying, respiratory infections, or fever from other causes. When symptoms continued beyond the newborn period, difficult nursing and poor weight gain were noted.

On examination of the chest there were dullness or tympany on percussion and diminished or absent breath sounds at the base of the chest posteriorly on the affected side, depending on the organs immediately under the diaphragm. Bowel sounds were heard in the chest in one infant in whom loops of intestine were in the thorax on the side of the eventration. Increased inspiratory divergence of the costal margin from the midline due to the unopposed action of the intercostal muscles (Hoover's sign) was noted in one infant. The signs of displacement of the heart to the opposite side were present.

The signs and symptoms of congenital eventration and hernia of the diaphragm may be exactly alike, although usually not so severe in eventration as in diaphragmatic hernia. The roentgenologic and fluoroscopic examinations furnish the only means of differentiation, but most often the differential diagnosis is established at operation. The roentgenologic finding of a high position of the diaphragm presenting as an unbroken curved convex line from the lateral wall of the chest to the mediastinum, with the abdominal contents beneath, is usually diagnostic of eventration. In congenital diaphragmatic hernia, the diaphragm is difficult to outline, and the portion that is visible is not elevated. The irregularity in density in the thorax on the affected side, the pattern of which suggests gas-filled loops of intestine, and the displacement of the heart and mediastinum to the contralateral side are noted in both congenital eventration and hernia of the diaphragm.

Paralysis of the diaphragm in the newborn child, producing respiratory difficulty due to phrenic nerve injury, may simulate congenital eventration of that organ (Schifrin[8]). This condition may or may not be associated with brachial plexus paralysis. Well-defined paradoxic movement of the elevated diaphragm on fluoroscopic examination of the chest is indicative of a paralysis of the phrenic nerve. The movement of the diaphragm is usually diminished in congenital eventration.

As in congenital hernia of the diaphragm, congenital eventration of that structure is more common on the left side than on the right. In the eight infants treated by operation, five were on the left side and three on the right. The strength of the affected side of the diaphragm is considerably reduced, and the diaphragm is elevated by the intra-abdominal pressure. The result on the left side is elevation in varying combinations of the stomach, the splenic flexure of the colon, the small intestine, the spleen, and the left lobe of the liver, and on the right side, elevation of the right lobe of the liver. The intra-abdominal viscera in the thorax displace the heart and mediastinal structures to the opposite side.

It is difficult to explain the difference in the structure of the diaphragm in the eight infants with congenital eventration treated by operation. In the right-sided cases, the diaphragm, although high in position, seemed to be normally developed with muscular tissue, whereas in the left-sided cases the diaphragm appeared as a thin translucent membrane.

The treatment of congenital eventration of the diaphragm is determined by the severity of the symptoms. Prompt surgical treatment should be carried out if dyspnea and cyanosis are severe. This has become the accepted practice in congenital hernia of the diaphragm, with a resultant marked reduction in mortality, particularly in the newborn period. Before transportation to the operating room, inhalation of oxygen should be supplied through an incubator or cubicle, the stomach emptied through a Levin tube, and the necessary fluids administered through a cut-down intravenous infusion in an ankle vein. The latter is utilized for transfusion of blood during operation.

Positive pressure anesthesia is helpful and is given through a small closed system using a tightly fitting face mask.

In the eight infants treated surgically, the operative technique consisted essentially in pulling the diaphragm down to the level of the margin of the diaphragmatic attachments, with or without excision of a portion of the diaphragm, and plication of the diaphragm with superimposed rows of interrupted mattress sutures of silk. In one infant, a method of tenting the diaphragm caudadward was used. Following these procedures, the newly constructed diaphragm was fixed to the thoracic wall, crus of the diaphragm, and renal fascia. The operative approach was thoracic in four infants, abdominal in three, and thoracoabdominal in one.

There was no operative mortality, and cure of the condition was effected in the recorded instances of congenital eventration of the diaphragm in infants treated surgically.

SUMMARY

The pathologic and clinical features, diagnosis, and treatment of congenital eventration of the diaphragm in infancy are reviewed.

An operative cure of congenital eventration of the diaphragm in an 8-day-old infant, the youngest recorded patient, is reported.

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BRONCHIOGENIC CYST ASSOCIATED WITH AN ANOMALOUS PULMONARY ARTERY ARISING FROM THE THORACIC AORTA

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ANOMALOUS pulmonary arteries were first described by Huber[1] in 1777. Until the relatively recent advent of pulmonary surgery, these aberrant vessels were only post-mortem curiosities. Up to the present time, there have been forty-two cases of an aberrant artery to the lower lobe of the lung reported in the literaure.[2, 3] The first report was made by Harris[4] in 1940; since then, Bruer, Clagett, and MacDonald,[5] Finley and Maier,[6] Tosatti and Gravel,[7] and Pryce, Sellors, and Blair[8] have all reported cases with their clinical summaries.

In 1946, Pryce and associates[8] published a comprehensive description of this congenital lung anomaly which was described as consisting of two components: part 1, the maldevelopment of a portion of the bronchial tree, and part 2, the anomalous artery from the aorta to the affected portion of the lung.

The anomaly may affect either of the lower lobes but has been found more frequently on the left, usually involving the posteromedial portion of this lobe.

Pryce's[8] hypothesis concerning this anomaly is quoted as follows: "A persistent artery from the dorsal aorta, instead of the ventral aorta, to the pulmonary anlage, attaches itself to one of the bulbous growing tips of the budding bronchi and then enters into competition with the developing pulmonary artery. As the embryo grows and straightens, and as the lung shifts to a more cephalad position, the captive bulb is gradually torn away and isolated (sequestered) producing rests from which cysts may develop. The anomalous artery usually has many of the physical characteristics of a pulmonary artery and these render it a relatively fragile vessel to carry blood at a systemic pressure."

CASE REPORT

R. C., a Negro boy, aged 6 years, was admitted to the Metropolitan Hospital on Nov. 28, 1952, for the repair of an umbilical hernia.

The mother furnished the past history that the child had been admitted to another hospital at 6 months of age for "difficulty in breathing" of four months' duration. A report from this hospital showed that the infant had an unexplained atelectasis of the left upper lobe which cleared after six weeks of hospitalization. A definite etiological diagnosis was not made, but there was complete re-expansion of the lung prior to discharge.

The physical examination at Metropolitan Hospital was negative except for a large, easily reducible umbilical hernia and several small, hard, discrete submaxillary, axillary, and inguinal lymph nodes. The hemogram revealed a hemoglobin of 11.8 Gm., 4,670,000 red blood cells, and 7,900 leukocytes with a normal differential count. Urinalysis and sedimentation rate were normal. On a routine roentgenogram of the chest (Fig. 1), a round

Received for publication, Sept. 17, 1953.

Fig. 1.—A rounded mass is visualized at the level of the left ninth rib in the paravertebral region.

Fig. 2.—Operative exposure demonstrating the anomalous artery entering the lung.

Fig. 3.

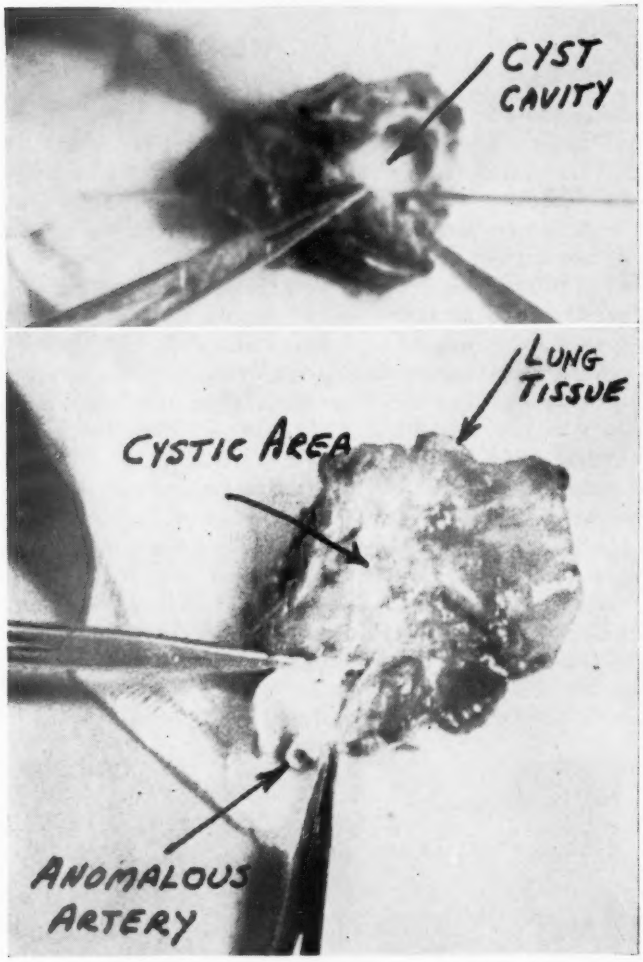

Fig. 4.

Fig. 3.—The operative specimen opened, demonstrating the cystic cavity.
Fig. 4.—Another view of the operative specimen, demonstrating the anomalous artery.

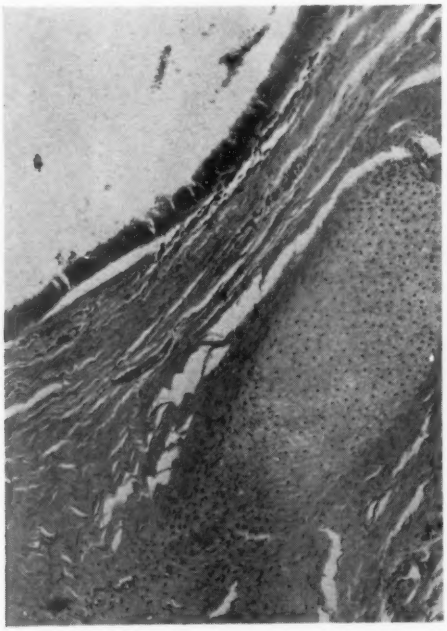

Fig. 5.—A photomicrograph showing a section of the cyst wall lined by ciliated columnar epithelium, fibrous tissue, and cartilage.

"quarter-sized" dense shadow was seen in the left lower lung field at the level of the ninth posterior rib. Lateral and oblique films showed that the shadow was posterior.

Because of this incidental finding of a lesion in the left chest, surgery for the hernia was postponed. The diagnosis of a posterior mediastinal tumor, probably neurogenic in origin, was considered. Operation for excision of this tumor was advised because of the known malignant potentiality of neurogenic tumors.[9]

Operation was performed on Dec. 29, 1952, under endotracheal anesthesia. Because of the preoperative diagnosis of posterior mediastinal tumor, an extrapleural approach was used. An oblique incision was made over the ninth rib, and a short posterior segment of this rib was removed subperiosteally. Since no tumor mass was found in the posterior mediastinum, the incision was lengthened, a longer segment of the ninth rib was removed, and the pleural cavity was entered through this rib bed. The posterior aspect of the left lower lobe was found adherent to the chest wall by thin bands of adhesions. These were separated, and an artery, approximately 6 mm. in diameter, was found arising from the anterior side of the aorta just above the diaphragm (Fig. 2). This artery coursed to the lung and entered the lower lobe on the lateral aspect of the posterior basal segment. The mass which had produced the abnormal x-ray shadow was found in this portion of the lung. It was rounded, walnut-sized, and rubbery in consistency. The anomalous artery was temporarily occluded, and as it produced no untoward symptoms, it was ligated and divided. A wedge resection of the portion of lung containing the tumor was then performed. On sectioning of the specimen, it was found to consist of a cyst containing thick, creamy, odorless material. Culture of this material later proved sterile.

Fig. 6.—The anomalous artery in cross section.

The postoperative course was uneventful except for gastric distention, which was relieved by suction. There was complete re-expansion of the lung. The underwater catheter drain was removed in twenty-four hours. The incision healed solidly. The patient was discharged after fifteen days with instructions to return one month later for repair of the umbilical hernia.

The pathologic report was as follows: On gross examination the specimen was a section of lung tissue which measured 6.5 by 5 by 3 cm., containing in its center a cystic

area 2 cm. in diameter (Fig. 3). This was lined by a smooth grayish white membrane containing odorless inspissated material. Surrounding the cyst there were several large blood vessels which were tributaries of an artery 6 mm. in diameter which entered the parenchyma at one end of the specimen (Fig. 4).

Microscopic examination showed the cyst to be lined by columnar ciliated epithelium of respiratory type (Fig. 5). The cystic cavity was surrounded by fairly dense fibrous tissue. Cartilage was also present. The vessel showed all the coats of a large-sized artery (Fig. 6).

SUMMARY

A case of bronchiogenic cyst associated with an anomalous artery has been presented. The hypothesis for the origin of this anomaly is quoted from Pryce.[8] This case differs from Pryce's description in that the anomalous artery actually had the characteristics of a systemic rather than those of a pulmonary vessel. It also differs from all of the case reports reviewed[2-8, 10] in that this patient had no respiratory symptoms, except in infancy, whereas the cases from the literature all refer to the symptomatology of chronic cough, sputum, frank pus, and hemoptysis.

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Editorial

A Note on Spinal Anesthesia and Its Relationship to a Larger Problem

THESE observations deal with a nonmedical limitation of spinal anesthesia and are concerned more with a principle underlying the practice of medicine as a whole than with spinal anesthesia. In one sense, my comments merely report a skirmish in a brush-filled hollow, but their implications relate to a larger and noisier battle now in progress.

At the moment I am not going to speak of the arguments between those who find spinal anesthesia useful and valuable and those who say it is akin to malpractice.

In this instance I can point to two items—and one seems to spring from the other—which are pertinent to the immediate subject: The first is an article written three years ago by the late Dr. Foster Kennedy and his associates.[1] In that article, which had wide coverage in the lay press, Dr. Kennedy, then near the end of a long and distinguished career in neurology, described twelve tragic cases of injury resulting from spinal anesthesia. Rebuttal to this was made:[2] ". . . to say that bad things can follow spinal anesthesia is to state the truth. What we all want to know is how often accidents occur (with spinal in comparison with other technics) . . . we are left in the dark as to how many spinal anesthesias were in the pool from which these 12 serious complications drained." It was pointed out that an equal number of serious neurological complications as a consequence of open-drop ether could be assembled.

Notwithstanding what seemed to me to be a fundamental defect in the Kennedy article (the failure to provide data on incidence of the accidents reported), it unquestionably had great influence. Some institutions promptly considered barring spinal anesthesia altogether. A recent extensive survey[3] of all the anesthesias in ten university hospitals scattered over the entire country, and made during a five-year period, demonstrated the surprising fact that spinal anesthesia accounts in these hospitals for less than 9 per cent of all anesthesias and is still declining in use. (Incidentally, in the same study, the death rate from spinal anesthesia appears to be low. It is impossible to consider this in direct comparison with other techniques, for the spinal technique is not generally used for difficult thoracic surgery, for example, which accounts for some of the bad news with other procedures.)

So much for the first point and the decline in use of spinal anesthesia. The second point is that insurance companies are, in some regions, refusing to protect physicians against damage suits when spinal anesthesia is employed. This brings us to the crux of these remarks: Are the techniques employed in the practice of medicine to be determined by competent physicians themselves,

or are outside pressure groups going to do it? If spinal anesthesia is not useful in proportion to its dangers, it should be eliminated by those skilled in its use. (Many, including myself, believe it to be valuable and important for certain purposes.)

Not long ago, a judgment of $160,000 was awarded and collected in the case of a patient who had been injured by spinal anesthesia, although considerable recovery of function had occurred in that case. Clearly, no individual physician can face such a threat with equanimity. This financial hazard can be accepted only if the institutions involved assume liability in the case of suit, while the medical profession itself decides whether it will retain or reject the technique of spinal anesthesia. The alternative is to allow insurance companies to determine the nature of medical practice in this instance, and thus allow further encroachment on the freedom of the physician.

—*Henry K. Beecher*

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Recent Advances in Surgery

Conducted by Alfred Blalock, M.D.

CARCINOMA OF THE ESOPHAGUS: A NEW CONCEPT IN THERAPY

Sixty Collected Cases, Using the Polyethylene Tube; Report of Ten (Selected)

Edgar F. Berman, M.D., Baltimore, Md.

(From the Department of Surgery, Sinai Hospital)

IN BROAD perspective, the results of esophageal surgery for malignancy do not lead to undue optimism. In the past two decades, the progress which has been made in the attempted cure of this disease can be attributed primarily to the newer adjuvant measures generally applicable to surgery; that is, modern anesthesia, antibiotics, and multiple transfusions, and the now well-grounded basic principles of thoracic surgery.

Some of the reports of large series which have been in the literature recently have been extremely discouraging. Parker[1] and his associates have had 170 cases of cancer of the esophagus, of which 114 were inoperable. Of the fifty-six which were operable, only thirty were resectable. The operative mortality averaged 60 per cent, and only three patients were alive at ten months, with one living for three years. There have been no recent five-year cures reported at The Johns Hopkins Hospital, according to Ravitch.[2] Sweet[3] reported 254 cases of carcinoma of the esophagus and cardia in a twelve-year period ending Jan. 1, 1951. The operative mortality in his hands was 24.3 per cent for midthoracic lesions. Of the total (107) of midthoracic patients operated on, only one survived five years. The number of five-year cures in the literature is relatively negligible.

A situation such as this calls for a thorough reappraisal of both disease and method. First, in the evaluation, two factors must be made clear; one is that carcinomas around the cardia and lower portion of the esophagus are in essence slow-growing adenocarcinomas, in contradistinction to malignancies above this point, which are extremely swiftly growing epidermoid carcinomas. Humphreys[4] deems this distinction necessary in any statistical analysis of longevity. Another factor which must be considered is that reports of large series usually come from medical centers with a high degree of specialization and experience with esophageal surgery. In reckoning mortality, it must be understood that every general surgeon does resect one or two cases of esophageal carcinoma each year, and from personal inquiry at smaller general hospitals, I find that the true mortality is well above the ghastly aforementioned figures.

Received for publication, Oct. 9, 1953.

We have reached our zenith in the techniques commonly used, but our results have not paralleled our technical facility.

Every portion of the esophagus has now been rendered available to surgical attack. The most popular method of maintaining continuity has been the esophagogastrostomy. Esophagojejunostomy and the antithoracic procedures have been almost generally abandoned for obvious reasons. The extent of the procedure of choice has grown to the point where the stomach may now be anastomosed to the esophagus in the neck. However, the rationality of this extensive procedure must be questioned, for the increase in extent of resection has been only in a vertical fashion. In a disease which seems to spread as rapidly laterally (both nodal and direct extension) as it does longitudinally, it is sheer delusion to suppose that our results will better with the increased length of esophagus resected. With a disease which frequently extends beyond its natural boundaries before symptoms occur, in a location which prohibits block or complete nodal dissection, it is extremely dubious that our present methods will better our present results.

It is highly improbable from the foregoing that the progress in this disease has been real. Concerning the postoperative course of the conventional procedure, the morbidity has been excessive. The number of leaks with empyema and fistulas are entirely too high for practicality. Esophagitis, ulcers, and strictures at the anastomotic site are not unusual before the final demise, because in this procedure the cardiac sphincter is obviated, with the anastomosis being constantly bathed in gastric juice. Obstructive recurrent malignancies at or near the anastomotic site are definite potential hazards. The vagotomized stomach in the chest predisposes to pulmonary complications.

In this disease, if most patients are to live only one year or two, one of the prime requisites of an operation should be a smooth posthospital course. However, even if the patient overcomes the hazard of morbid complication, he is usually still uncomfortable with the stomach in the chest. The quantity of food to be taken at each meal is restricted. There is usually postprandial dyspnea and palpitation. There is acid regurgitation with substernal burning and a frequent feeling of fullness in the chest.

With these facts before us, it can readily be seen that the present-day operative procedures have failed to prove themselves in any way in a trial period of over a decade and a half. The mortality is still tremendous, the longevity short, the morbidity excessive, and the comfort of the patient minimal. The procedure has taken on the aspect of a tour de force. However, any surgical tour de force for malignancy is valid only when a modicum of good is done or when the method by constant usage may lead to variations and embellishments to a procedure which will be in the realm of practicality as to figures and comfort. This has not been the case.

The procedure which we deem more rational for this disease, namely, the substitution of a plastic tube for the resected portion of the esophagus, is offered only in the light that the same extent of resection can be carried out with a greatly decreased operative mortality and morbidity, the same longevity, and advantages to the patient of comfort and normal eating. We feel,

after a short collected experience comprising some sixty cases, that the "plastic esophagus" is the temporal answer to the question of procedure in malignancies of this organ. The following data substantiate our feelings.

Material and Experimental Work.—The polyethylene plastic is extremely tissue-compatible with negative water absorption, is nonporous, and easily sterilized. The tubes are unchanged when buried in human tissue. There is no foreign body reaction. They conduct heat poorly, and durability under ordinary body conditions has been very satisfactory. The tube may be left in permanently. Substituting this tube (Fig. 1) by the simple technique which I previously described for resected portions of esophagus has shown that a new fibrous sheath forms, in both animals and human beings, around the tube, which eventually (sixty to ninety days) becomes epithelized, giving a new esophagus without musculature.[5-7] This lack of musculature does not impede the flow of ingested material, as will be shown in Case 1 (previously reported), where the tube was removed by esophagoscopy after one year. Extracting the tube is not advised in cases of malignancy.

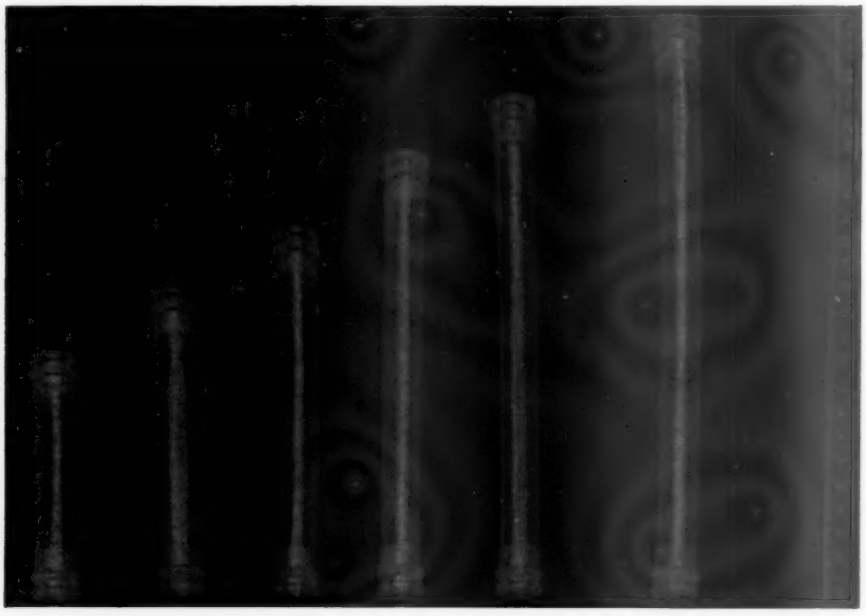


Fig. 1.—The set of six polyethylene tubes used, measuring from four to nine inches in length. After resection of the lesion, the tube is approximated for length, making sure that the full cuff is covered with esophagus. Every size should be available at each procedure.

Technique (Fig. 2).—The simplicity of this procedure allows any surgeon familiar with the thorax to perform the operation without undue risk. This is an extremely important consideration. The right chest alone is utilized for the approach, with no disturbance of the diaphragm or other intra-abdominal structures. It is unnecessary to extend the incision into the abdomen, for if there is intra-abdominal extension, the procedure is simply palliative. The aorta does not impede. There are no time-consuming, minute anastomoses necessary. There is no question of suture-line tensions. As much of the

esophagus as is necessary (from the cervical region to the stomach) (Cases 1, 2, 3, and 4) may be resected, just substituting a longer tube. No case is inoperable, for when extension of the tumor has occurred, the primary growth may be removed, leaving some malignancy on the surrounding vital structures, but replacing the defect with a tube. The operating time for these elderly malnourished patients is approximately one and one-half to two hours. This procedure is not carried out with palliative intent, and should be as curative as any other procedure for the disease. A newer,* safer, and simple technique with which we are working will be presented soon as an alternate to the original one.

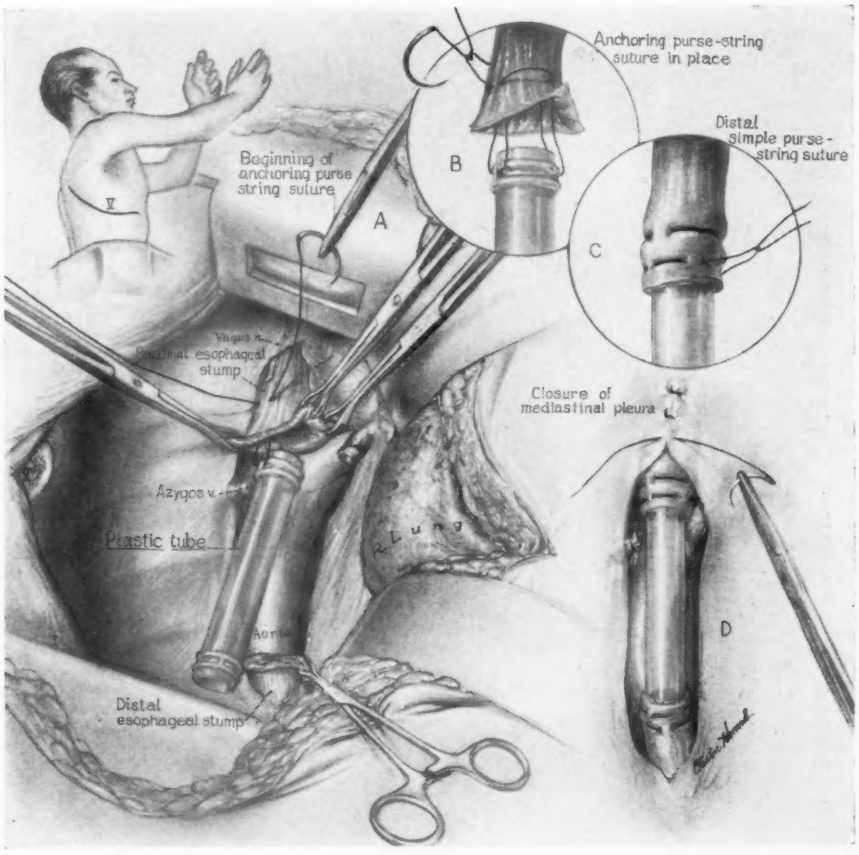


Fig. 2.

Mortality.—In the collected sixty cases, there has been less than 10 per cent operative mortality. All cases which I handled are deemed operable. There were five deaths. There is no shocking devascularization and mobilization of the stomach. There is no great blood replacement necessary with concomitant disturbances in hemodynamics. The deaths that we have had within the first four weeks postoperatively have been from coronary thrombosis, massive pleural effusion, atelectasis, cerebral hemorrhage, and one empyema.

*By August, 1953, only the newer tubes will be on the market. These will be distributed by the Murray-Baumgartner Surgical Instrument Co., 5 West Chase Street, Baltimore, Md.

A. *B.* *C.*

Fig. 3 (Case 1).—*A*, Preoperative barium swallow showing a defect in the lower end of the esophagus measuring 3 cm. in length. *B*, Six months postoperatively the continuity of the barium flowing through the tube and stomach is shown. *C*, Twelve months postoperatively barium swallow shows the ability of the new esophagus formed around the plastic tube after the tube had been removed by esophagoscopy.

Morbidity.—It is inconceivable that any untoward pulmonary complications would be expected when the mediastinum is closed tightly over this plastic prosthesis, which is of the same diameter as the normal esophagus. In the sixty cases reported, there have been three leaks which have closed within one week, and one empyema which was fatal. There were two cases of loculated fluid collections cured by antibiotics and aspiration. There have been no mediastinal abscesses. One tube slipped into the stomach postoperatively and was retrieved.

Longevity and Comfort.—We do not expect these patients to live any longer than the average patient treated by other methods. Having handled these cases for the past two and one-half years, our longest survival has been twenty months, with maximum comfort. These have been no symptoms of acid regurgitation, stricture, or esophageal ulceration. Three patients have died of metastases. At post-mortem, two showed the tube entirely encased by epithelized fibrous tissue; one was encased in tumor tissue with no obstruction. Every patient has eaten normal meals perfectly well and comfortably until his demise.

CASE REPORTS

CASE 1.—I have previously reported this case in detail. A. G., aged 75 years, was admitted with carcinoma of the lower end of the esophagus, and had a resection of the lower 6 cm. of the esophagus and the uppermost portion of the stomach. A plastic tube, 12 cm. in length, was inserted. At operation, lymph node metastasis was present. The patient ate comfortably and lived for thirteen months. One month before his demise, the tube was extracted with an esophagoscope, and he had no difficulty in eating after this. A post-mortem was not obtained. The cause of death was generalized metastases (Fig. 3).

CASE 2.—E. C., aged 46 years, was admitted to the hospital with a complaint of dysphagia and regurgitation, with substernal burning for six months. On admission, the patient had lost thirty pounds in weight. He complained of pain in the right elbow, which later turned out to be a metastatic lesion from the esophagus. X-ray film and biopsy confirmed the diagnosis of squamous cell carcinoma of the esophagus. At operation, a lesion, approximately 6 cm. in length, was found with lymph node metastasis. The lower end of the esophagus and the cardia of the stomach were resected, pulling the stomach up through the hiatus, and a six-inch plastic tube was inserted. Postoperatively, the patient had a somewhat stormy course for a few days with a left lower lobe pneumonia, but was discharged one month postoperatively, eating practically a normal diet. He went back to work two months postoperatively. The patient had no complaints, but gradually lost weight and died of generalized metastases, corroborated by post-mortem thirteen months postoperatively. Post-mortem revealed the plastic tube lying loose but snug within an entirely new epithelial sheath (Fig. 4).

Comment.—An interesting point in this case was symptomatic evidence of metastasis of the right elbow at the time of the original symptoms.

CASE 3.—J. P., aged 65 years, was admitted to the hospital with a history of dysphagia of one year's duration and forty-pound weight loss. X-ray film revealed a lesion in the mid-portion of the esophagus, confirmed by biopsy. At operation, a 7 cm. mass tightly adherent to the aorta and right main-stem bronchus was resected with accompanying lymph nodes. A seven-inch plastic tube was inserted. This patient's convalescence was extremely smooth, and he was discharged in two and one-half weeks. He is still living and well, and eating perfectly normally (Fig. 5).

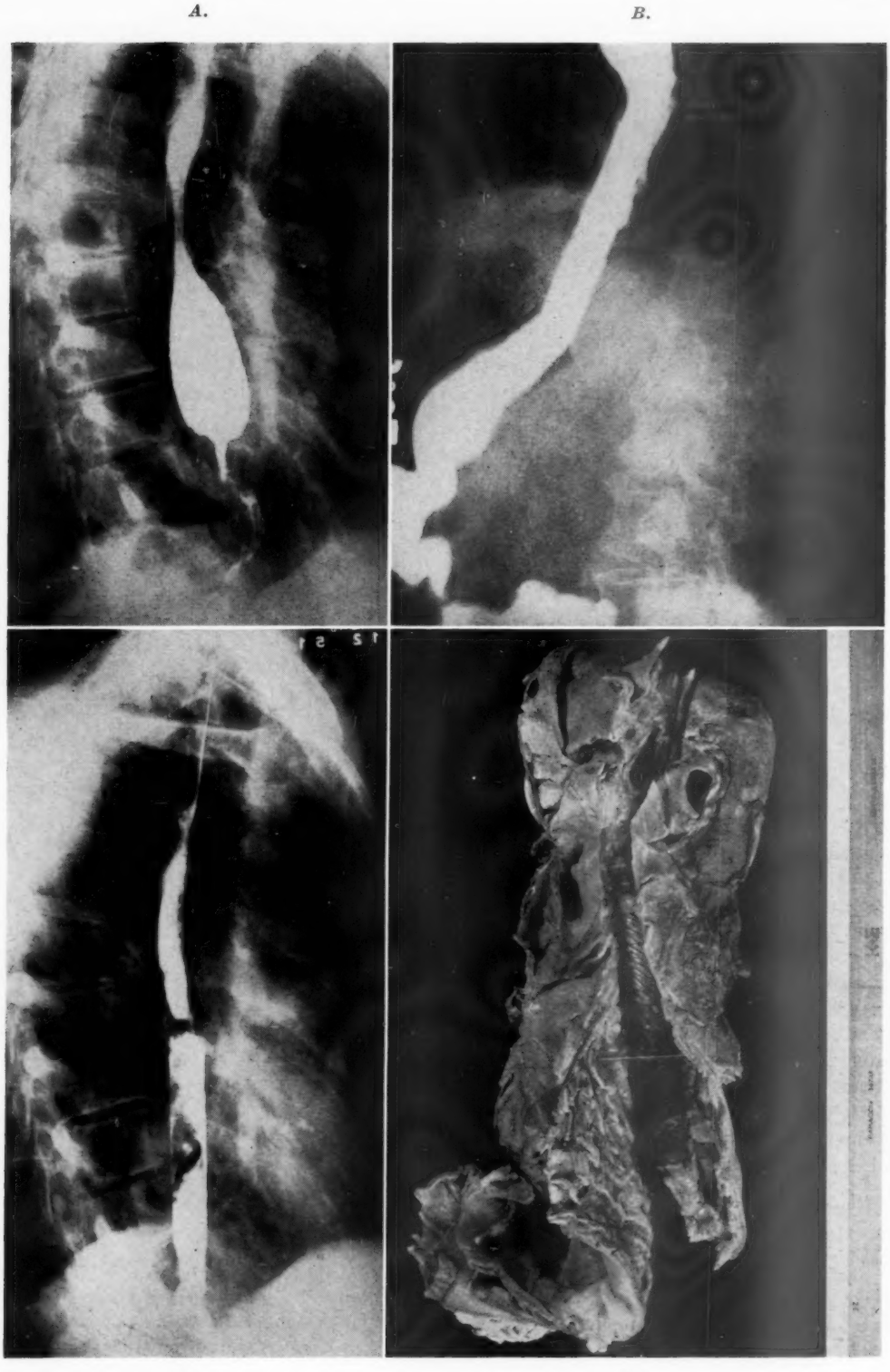
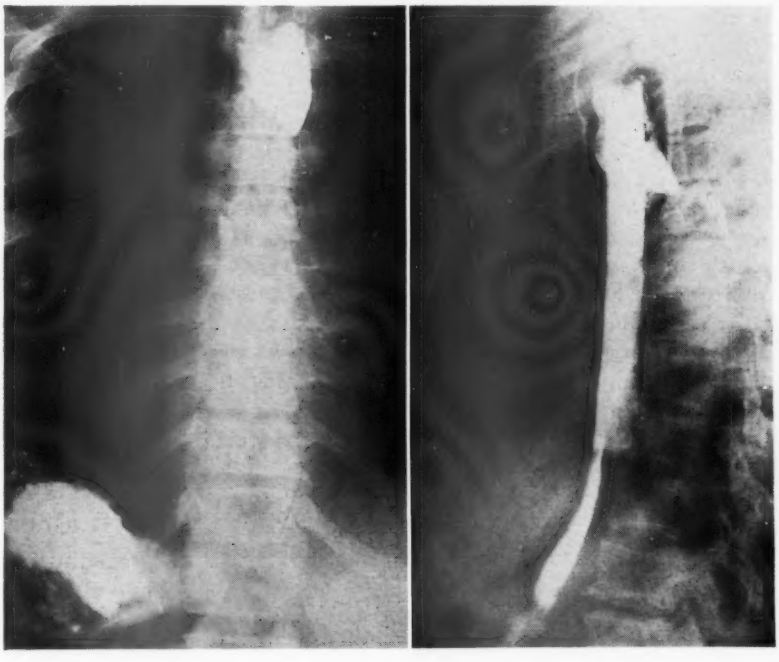


Fig. 4 (Case 2).—*A*, The lesion is shown at the lower end of the esophagus. *B*, Fourteen days postoperatively, barium is seen going through the proximal esophagus, the tube, and into the stomach. *C*, Barium swallow, seven months postoperatively. *D*, At post-mortem ten months postoperatively, the tube is seen completely surrounded by fibrous tissue; however, there was no obstruction.

A. *B.*

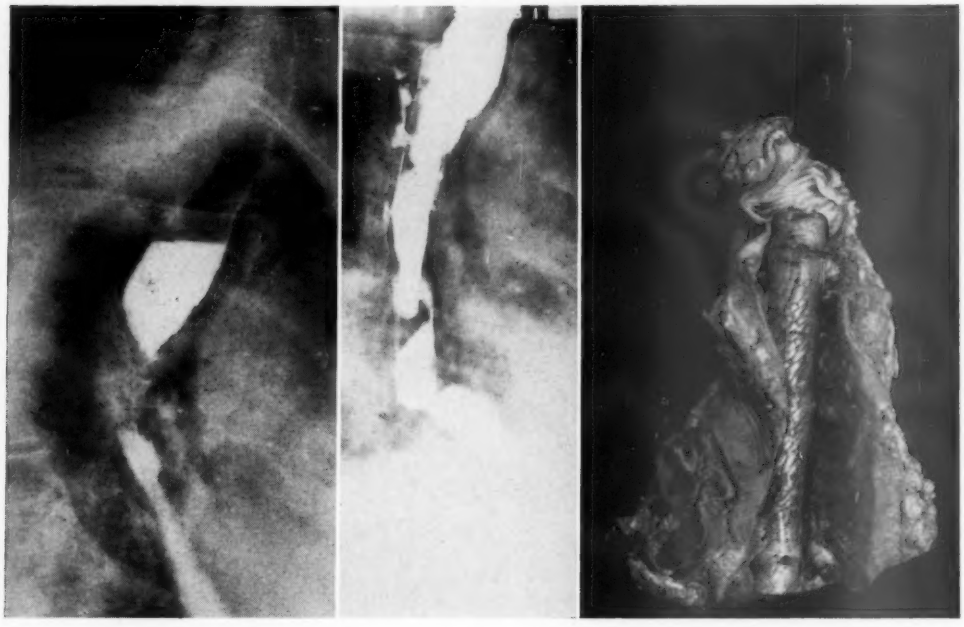
Fig. 5 (Case 3).—*A*, The malignant obstruction in the mid-esophagus. *B*, The polyethylene tube is shown in place three weeks after resection.



A. *B.*

Fig. 6 (Case 4).—*A*, A high malignant obstruction is seen in the esophagus. *B*, The barium-filled tube is shown with some of the opaque material around the tube but within the new sheath.

CASE 4.—R. S., aged 49 years, was admitted with dysphagia and "food sticking in his throat" with no relief without vomiting. Esophagoscopy showed a squamous cell carcinoma in the upper middle third of the esophagus. At operation, the malignancy involved the left main-stem bronchus, some of which was left attached. A seven-inch plastic tube was inserted. The patient was discharged from the hospital in fifteen days with an excellent follow-up five months postoperatively (Fig. 6).



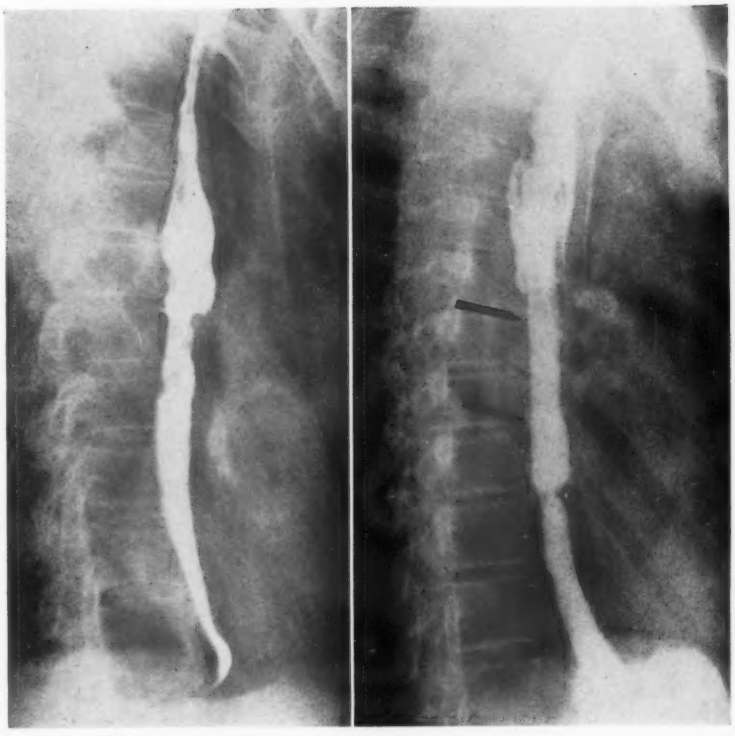
A. *B.* *C.*

Fig. 7 (Case 5).—*A*, Roentgenogram revealing an obstructive lesion in the upper one-third of the thoracic esophagus in a patient with an extreme dorsal kyphosis. *B*, Postoperative barium swallow shows the tube conforming to the curvature of the spine with barium passing through. *C*, Seven months postoperatively post-mortem shows the tube completely surrounded by fibrous tissue.

CASE 5.—C. W., a 66-year-old physician, was admitted with a history of dysphagia for four months following a total laryngectomy for carcinoma. Barium swallow and biopsy revealed a squamous cell carcinoma of the middle third of the esophagus. The patient had an extreme dorsal kyphosis. At operation, a carcinoma, measuring 5 cm. and adherent to both the carina and aorta, was found with regional metastases. This was resected, leaving a portion of malignancy on the aorta, and a seven-inch plastic tube was inserted. The patient received irradiation to the area of resection postoperatively. He had a relatively smooth postoperative course and went back to his practice. Approximately seven months postoperatively, the patient had a severe hemorrhage from the lung, and post-mortem revealed that the carcinoma of the esophagus had ruptured into the main-stem bronchus.

Comment.—This case was interesting in that it was possible for the patient to speak as clearly as before by the esophageal voice method. It is doubtful that he would have had speech with a high stomach anastomosis. Second, the perforation was probably abetted by the postoperative x-ray therapy to the site of lesion (Fig. 7).

CASE 6.—J. S., a 70-year-old man, had a carcinoma of the upper end of the middle third of the esophagus, which was resected, and a 14 cm. tube was inserted. The patient was dis-

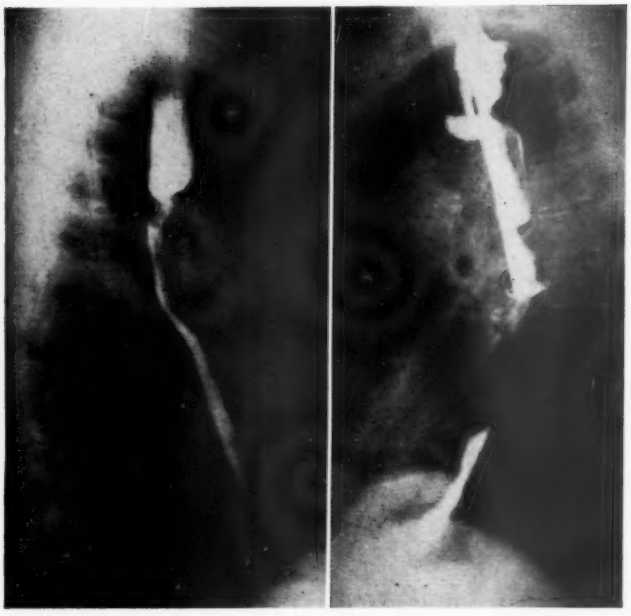


A. *B.*

Fig. 8 (Case 6).—*A*, Carcinoma of the midthoracic esophagus is easily seen. *B*, Five weeks postoperatively, the plastic tube is seen carrying the barium down to the stomach.

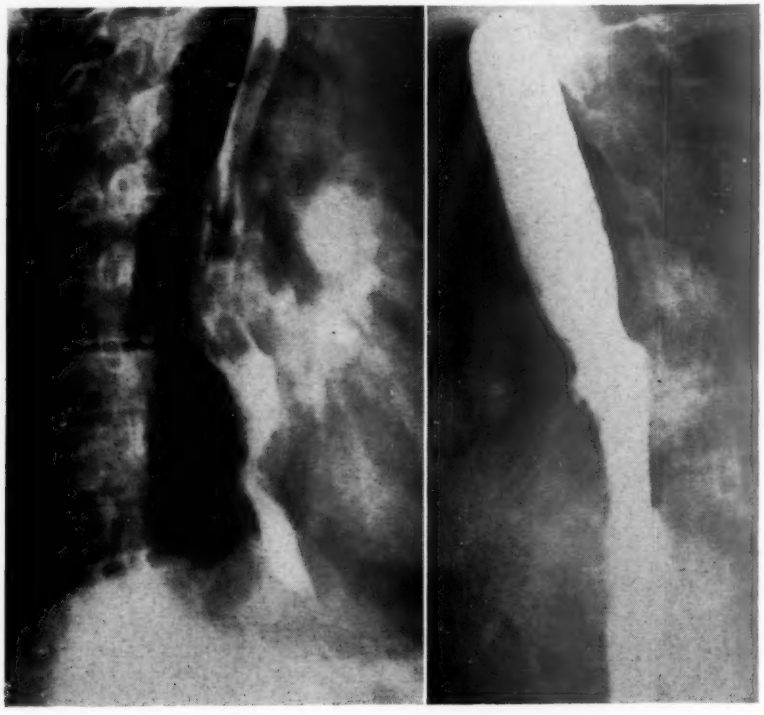
A. *B.*

Fig. 9 (Case 7).—*A*, Squamous carcinoma of the upper end of the lower one-third of the esophagus. *B*, Two weeks postoperatively the barium is seen passing uninterrupted into the stomach. The rim of the upper mouth of the tube is seen in this film.



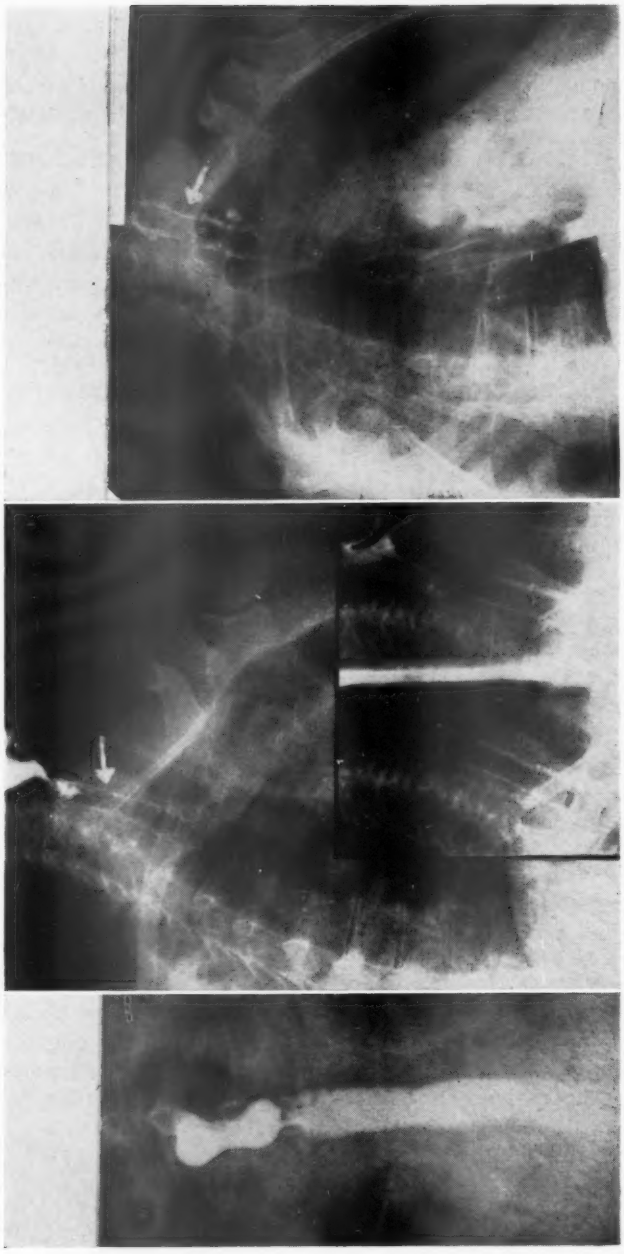
A. *B.*

Fig. 10 (Case 8).—*A*, A large infiltrative squamous lesion in the middle one-third of the esophagus. *B*, Barium shown going readily through the tube and into the stomach. Much malignancy was left on the surrounding structures in this case. Barium may be seen outside the tube but within the new sheath enveloping the tube.



A. *B.*

Fig. 11 (Case 9).—*A*, The lesion seen extended from above the azygos vein down to the cardia. *B*, The barium is shown going through the nine-inch tube postoperatively. The lower anastomosis is to the stomach.



A. *B.* *C.*

Fig 12 (Case 10).—*A*, Initial lesion is nonradiographic. This film denotes spasm at the site of biopsy in the cervical region. *B*, Six weeks postoperatively, the barium is shown going through the plastic tube into the thoracic esophagus (with insert). *C*, Lipiodol swallow ten days postoperatively shows the tube and the anastomotic areas.

charged eating practically a full diet, and is still living twelve months postoperatively (Fig. 8).

CASE 7.—C. C., a 52-year-old white woman, was admitted with dysphagia and symptoms of substernal obstruction for a period of eight months. Biopsy revealed squamous cell carcinoma of the upper end of the lower third of the esophagus. An exploratory laparotomy showed widespread abdominal metastasis. A thoracotomy revealed a large extensively invasive carcinoma, which was removed, and a seven-inch plastic tube was inserted. The patient is still living and eating well five months postoperatively.

CASE 8.—G. H., aged 52 years, was admitted to The Johns Hopkins Hospital with a complaint of substernal discomfort while eating coarse foods. This progressed to dysphagia and regurgitation and a ten-pound weight loss. At this time, it was felt by the surgeon that the patient was inoperable, and a first stage antithoracic jejunal loop was brought up with a gastrostomy. The loop became nonviable. The jejunal loop was taken down and the chest wall drained. Two weeks later, the patient was again taken to the operating room, and a large infiltrative lesion with definite evidence of metastases in every direction was removed at about the fourth dorsal vertebra, leaving malignancy behind. He ate well on the seventh postoperative day and was discharged eighteen days postoperatively. There was no follow-up.

Comment.—This case shows that even in the most extensive lesion, and as a last resort, with the patient in an extremely bad condition, palliation is extremely simple with the tube method (Fig. 10).

CASE 9.—The history of A. D., aged 68 years, began seven months prior to admission with dysphagia and weight loss. Esophagogram revealed an extensive lesion of the middle and lower third, proved by biopsy to be carcinoma. This patient was quickly becoming worse at the time of operation, with nutrition being maintained solely by the intravenous route. At operation, a tremendous lesion was found extending from the azygos vein to the cardia, and it was resected. The hiatus was enlarged by finger dissertion, and a portion of the stomach was drawn through for the lower anastomosis. The patient did extremely well and was discharged on a regular diet. A four-week check revealed the tube to be functioning well, but the patient was still losing weight (Fig. 11).

CASE 10.—A. G., aged 52 years, complained that some months prior to admission he had swallowed a chicken bone which seemed to stick in his throat. Since that time he had had a sticking sensation in the neck, with some dysphagia. X-ray film was inconclusive, but esophagoscopy revealed a tiny 5 mm. lesion in the cervical esophagus which was biopsied and showed a squamous carcinoma. X-ray film (Fig. 12, *A*) shows the spasm at site of biopsy. At operation through a left cervical incision at The Johns Hopkins Hospital, the site of lesion was barely palpable, but a wide resection of the esophagus was performed, and a special (lesser diameter) three-inch plastic tube was anastomosed to either end of the esophagus. The patient has eaten perfectly well since then, with his only symptom being excessive salivation six months postoperatively.

SUMMARY

With the results as stated by different authors in large series, it seems that in concentrating our attack on the disease, the regard for the patient has become secondary. In the lapsed period of trial of almost two decades with all modern aids, a re-evaluation of the present methods of maintaining continuity after resection of portions of the esophagus for malignancy finds them sadly lacking. As reflected in the mortality, the conventional procedure is of too great a magnitude for the average patient in this group. The high mor-

bidity attests to the imperfections in the usual technique. The short survival makes us reflect on the fact that the intrinsic nature and the anatomic relationship of this disease make it not suitable for true cancer surgery. And, last, if the patient clings so tenaciously to life so as to overcome these obstacles, he deserves a better fate, in his score or so of remaining months, than the miserable one (if it is a mid or high lesion) that most of these patients are destined for.

The polyethylene prosthesis in our short collected experience seems to offer hope, but not for the principal factor, that of increased survival time with possibility of cure. Every surgeon familiar with thoracic technique can use this method with relative safety. However, a word of caution should be interjected. Although the success of this new and rather unconventional procedure requires no undue skill, the few fundamentals of the procedure, previously alluded to, should be adhered to, and some animal or cadaver rehearsal should be carried out before the first procedure. However, if used correctly, it will allow many more patients a few years' survival by dint of the low operative mortality. There will be a decreased and less hectic postoperative course, with fewer complications. Most important, to these unfortunates who are doomed, their remaining years will be comfortable, and they will have the ability to eat whatever and as much as they want with no concomitant distress.

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Book Reviews

Nash's Surgical Physiology. Revised and edited by Brian Blades, M.D., professor of surgery, George Washington University School of Medicine. Pp. 686. Springfield, 1953, Charles C Thomas, Publisher. $12.50.

The second edition of *Nash's Surgical Physiology* represents a considerable revision and edition by Brian Blades and his associates of the George Washington University School of Medicine of the earlier work. This new edition contains considerably more material presented in a more readable format along with an increased number of illustrations. Basically, the book deals with practical physiologic considerations as applied to clinical surgery. The material is grouped according to the major body systems, dealing successively with the cardiovascular system, burns and tissue repair, the respiratory system, the gastrointestinal system, the body fluids, the kidneys, the endocrine system, and the nervous system.

The effective stimulus of cardiac surgery to an increased interest in physiology by surgeons is reflected in this work by a chapter concerning recent advances in cardiovascular surgery. A similar expansion is seen in the treatment of physiology of the adrenal gland in the light of the recent tremendous expansion in this field. The authors make considerable reference to current articles on the subject matter.

As is inevitable when attempting to sort and organize the tremendous volume of physiologic data with reference to the equally enormous subject matter of general surgery, there is a tendency to present some controversial material in a dogmatic fashion as well as to retain a few outmoded concepts. Nevertheless, the authors have succeeded in covering a vast scope of material in a concise fashion without becoming entangled in a web of details.

While the book cannot be claimed to be a "royal road" to an easy and complete grasp of the subject matter (as, indeed, in this field no book can!), it does arrange the vast array of physiologic data along the path of interest of the clinical surgeon such that he may see it easily in respect to his field of interest.

This work should be of particular interest to the student, intern, and surgical resident, as well as to the practicing surgeon who wishes to refamiliarize himself with physiology in a broad sense as applied to the field of surgery.

Praktikum der Chirurgie. Ein Leitfaden für Studenten und Ärzte. By Prof. Dr. Med. Hans Hellner. Pp. 720. Munich-Berlin, 1952, Urban & Schwarzenberg.

This is the textbook of surgery written by the late O. Nordmann, revised for the seventh edition by Hellner. As in previous editions, the chief emphasis is laid on indications for surgical treatment, technique of emergency operations, pre- and postoperative care, and recognition of postoperative complications.

The book is written mainly from the personal experience of the authors, and no references are given. Since all the subspecialties of surgery are covered, treatment of many topics is necessarily brief. The surgery of heart disease is represented only by a short discussion of penetrating wounds and of pericarditis. Vagotomy is not mentioned in the section on treatment of peptic ulcer. The discussion of hernia repair takes no account of the newer knowledge of anatomy.

This volume is of interest as a reflection of current German practice, but it is of little value compared to the best books available in English.

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